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VIRGINIA MEDICAL MONTHLY

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GUEST EDITORIAL

Poliomyelitis

THIS time last year the State of Virginia was suffering from an epidemic of poliomyelitis which was requiring many hospital beds for treatment. In spite of the facilities present, there were many deaths.

Upon reviewing the outcome of last year's epidemic much was learned. Although very little more is known as to the spread of the disease some knowledge has been obtained as to treatment.

Early diagnosis, so that treatment may be started as soon as necessary, should save some lives.

The early symptoms are not pathognomonic for poliomyelitis and could be due to many other common illnesses, appearing during an epidemic of poliomyelitis. Such symptoms as fever, headache, and those related to the upper respiratory tract or gastrointestinal tract or to both may be common to many conditions. When meningeal irritation, spasm and weakness appear the diagnosis becomes simpler for poliomyelitis.

From observations of these cases suffering with the severest paralysis it has been noted that many of these patients refused to give in to their sickness. Instead of taking to bed or rest they exposed themselves to activity and stress without slowing down.

There has been the individual with fever, headache, and malaise who goes swimming and another who practices football. The mother who is caring for her child with poliomyelitis will often develop the disease to a greater extent.

From these cases and similar ones it appears that fatigue during the acute stage can be deleterious and that rest and the slowing down of patients' activities during the acute stage or immediately afterward is very important.

When a patient has developed paralysis and it appears to be progressing or when a patient has shown any signs of clouding of sensorium or irregular and strained breathing he should be sent to a hospital immediately for treatment.

Every physician should be aware of the possibility of the presence of poliomyelitis and be ready to make an early diagnosis so that he may advise his patients to go to bed if the disease is suspected. He should also encourage his patients to avoid excessive strain or fatigue during an epidemic. If these simple principles can be carried out the seriousness of poliomyelitis will be somewhat diminished.

LEE E. SUTTON, JR., *Chairman*
Committee on Poliomyelitis,
Medical Society of Virginia.

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LOWER NEPHRON NEPHROSIS*

M. MORRIS PINCKNEY, M.D., F.A.C.P.,
Richmond, Virginia.

A 29 year old woman in the eighth month of a supposedly normal pregnancy had severe labor pains and five hours later upon admission to the hospital was found to have a premature separation of the placenta, the fetus being dead. Delivery was effected and blood transfusion was administered.

Only a small amount of urine was voided in the first 24 hours of hospitalization, far too low in proportion to the intake of fluids, and urinalysis revealed albuminuria, blood and pus. Twenty-four hours after admission chemical studies of the blood revealed a high degree of nitrogen retention, an elevated icterus index and uric acid.

During the first five days of hospitalization measurement of her urine excretion showed oliguria throughout, but on the sixth day diuresis began and the patient made what is traditionally described as an uneventful recovery.

A slide exhibiting the chemical alterations in her blood is prepared from specimens obtained the day before the onset of diuresis.

Here we have an example, fortunately of happy outcome, of what Lucke[1] terms lower nephron nephrosis, a syndrome of vast importance and interest to clinicians in all fields, whether surgeons, physicians, obstetricians, or orthopedists, and one which pathologists especially in this country and England have elucidated most brilliantly. Lower nephron nephrosis may be observed clinically following a variety of injuries to the body, Lucke listing severe trauma to muscle (known as the crush syndrome), non-traumatic muscular ischemia, burns, transfusions with incompatible blood, heat stroke, black water fever, toxemias of pregnancy, uteroplacental damage, alkalosis, drug intoxications, and poisonings with certain vegetable and chemical agents. Whereas shock and renal failure are the clinical manifestations of these assaults upon the body, the pathologist discovers the anatomical common denominator in the lower segments of the nephrons.

The nephron, by definition, is an anatomical and functional unit of the kidney, being composed of the glomerulus, the proximal convoluted tubule, the descending loop of Henle, the ascending loop of Henle, and the distal convoluted tubule. The collecting tubule is not included in the unit.

Briefly, the glomerulus is a passive filtration plant, the filtrate draining into the proximal convoluted tubule whose large brush bordered cells initiate a process of active and selective reabsorption. Here 80 per cent of the fluid of the filtrate is reabsorbed and returned to the body's circulation, together with sugar and chlorides, according to the body's needs, and probably other chemical modifications not yet understood are performed. The narrowed descending limb of Henle's loop acts as a pressure trap prolonging contact between intratubular fluid and the resorbing surface of the proximal tubule.

The ascending loop of Henle and the distal convoluted tubule, the structures disordered in the lower nephron syndrome, perform the final modification of filtrate to urine, in particular by the selective resorption of chemicals to maintain the acid-base equilibrium of the blood stream. It is interesting to note[2] that each day more than a pound of the sodium salt is absorbed by the renal tubules to conserve base for the body.

The circulation of blood through the kidney is now being extensively restudied by physiologists and research clinicians. Less than three years ago Trueta[3] and his associates in England showed conclusively that there exist in the kidney of the rabbit two pathways of arterial circulation, rather than only one as had been previously thought. The main arterial channel, carrying about 85 per cent of the renal blood flow in the normal state, lies in the cortex, but a second course lies in the medulla and juxtamedullary areas of the cortex. This juxta-medullary shunt, therefore, when the main cortical channel is closed off, will carry the flow from the renal artery through the medulla to the renal veins, the cortex of the kidney being ischemic. In such a situation, only the juxta-medullary glomeruli, which are few in number, will receive blood.

In addition, another little understood part of the kidney is the so-called "juxtaglomerular appara-

*Read before the annual meeting of the Medical Society of Virginia at Roanoke, October 8-11, 1950.

tus", a collection of cells lying in the angle between the afferent and efferent arterioles of the glomerulus. These cells of possibly a neural or secretory character may regulate the glomerular blood flow by the liberation of renin.

A second shunt, in addition to Trueta's juxta-medullary channel, is the shunt described by Ludwig between the afferent arterioles of the glomeruli and the tubules. Thus there are two ways within the kidneys by which the glomeruli can be deprived of arterial blood—first, by a shunting of blood through the medulla by Trueta's juxta-medullary pathway leaving an ischemic cortex, and, second, by Ludwig's shunt by-passing the glomeruli between arterioli and tubules.

The mechanism of renal failure in the lower nephron syndrome has not been explained to the satisfaction of all investigators, but each writer agrees that "shock" is a constant finding in the early phases of this syndrome. Cournand[4] regards the production of shock as part of the homeostatic changes initiated by the body to protect its most vital parts from inadequate circulation. He feels that there is an active vasoconstriction of the kidney circulation, and suggests that the kidneys, like the skin, are temporarily cut off from the circulation, representing a manifestation of the body's determination to protect itself from death.

That this diminished renal circulation results from a shunting of blood through Trueta's juxta-medullary channels producing an ischemia of the renal cortex is postulated by Trueta and agreed to by many writers, according to Best and Taylor.[5] However, strong opposition to this theory is voiced by Lucke, Mallory,[6] and Phillips and Hamilton,[7] and Lauson[8] has demonstrated in man a marked diminution in the entire flow of blood through the kidney rather than any shunting mechanism. Furthermore, Maxwell, Breed and Smith,[9] only a month ago, published evidence to show that the "juxta-medullary circulation in man has no unique functional significance as far as can be ascertained at the present time".

If, therefore, Trueta's hypothesis of the cause of renal failure is rejected, what mechanism initiated by the state of shock produces the oliguria? Evidence has been presented by Phillips and Hamilton, and seemingly recently confirmed by Maxwell who states that "the more probable explanation appears to be that protracted anoxia or chemical injury destroys the integrity of the renal tubules leading, to a greater or lesser extent, to reabsorption of all constituents in the tubular urine".

Bywaters,[10] in his excellent article "Ischemic Muscle Necrosis", showed that in the crush syndrome the injured muscle quickly loses a large percentage of its pigment myoglobin, of its phosphorus, potassium, creatinine, and acid producing substances. These chemicals are dissipated on the first day after the injury and are rapidly excreted by the kidneys.

The chemical degeneration of myoglobin, the heme compound normally found in muscle, or of hemoglobin of the blood to simpler heme fractions may lead to precipitation of these compounds in the renal tubules, thus producing plugs of blood products to dam the tubules. Bywaters and Stead[11] have shown that blood pigment will be precipitated out of solution within the tubules in a tubular fluid of sufficient acidity, and this phenomenon in the presence of abnormal water reabsorption will lead to the formation of pigment plugs in the tubules. Pathologists, however, doubt that such tubular plugs cause oliguria, for microscopic examination of renal sections show only a few tubules so blocked.

Whether the flooding of the blood stream with potassium initiates either vascular constriction or in any way affects the formation of urine is undetermined, but future researches may clarify this point. It is known that a low blood sodium level will diminish renal blood flow and depress glomerular filtration, but in the lower nephron syndrome the frequent finding of low sodium values is believed to be the result and not the cause of the renal dysfunction. All we can say at present is that the blood electrolytes are probably not the initiating factors in the production of the kidney failure.

Clinically one sees a rapid diminution in the volume of urine, a few hundred c.c. or less being secreted in the second 24 hours after trauma. The first urine passed may be normal, having been formed before the injury, but after injury it becomes highly acid, low in specific gravity, and contains albumen, red cells, and pigment granules. Mallory states that even though the color may be normal that the benzidine test is usually positive, and that the color is rarely port wine except in transfusion reaction and crush cases. Total anuria may occur. The composition of the urine tends to resemble glomerular filtrate in that the concentration of urea is low and the chloride

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content is high. Potassium and creatine, derived from injured muscle, appear in the urine, being present in high concentrations in the blood.

With progressive renal decompensation comes nitrogen retention usually associated with low blood chlorides, lowered Co_2 combining power, and sometimes hypocalcemia. Lowering of the chlorides may be augmented by pre-renal deviation of these electrolytes through vomiting and perspiration.

The clinical picture of lower nephron syndrome, having been so frequently described,[12] will be sketched hurriedly. Following the injury to the body, injury due to crushing force, uteroplacental damage, transfusion reactions or what not, the patient goes into shock, mild or severe, lasting a few hours and this is the period in which renal damage is sustained.

Symptomatically the patient may show evidences of anxiety, and objectively the blood pressure and pulse may be normal, but if compensatory vasoconstriction is not maintained, the pressure falls, the systolic pressure falling more than the diastolic. Cournand finds that an accelerated pulse rate is inconstant and not a reliable criterion.

With the development of renal failure, the symptoms usual in this condition are found: apathy with periods of anxiety, confusion, edema, anorexia, vomiting, twitchings, convulsions, coma. Electrocardiographic evidences of potassium intoxication may be found:[13] high, narrow T waves, broadened QRS complexes, loss of P waves, and cardiac arrest.

The initial pathologic change[6] is seen 18 to 24 hours after injury and this is a lipid vacuolization of the ascending limb. Then after 24 to 72 hours one sees precipitation of pigment followed by moderate dilation of the proximal and distal convoluted tubules.

By the fourth or fifth day necrosis and regeneration of epithelium in the ascending limbs and distal tubules become evident. From the fifth day on one sees rupture of the tubules with herniation of their content into the stroma. There are no pathognomonic gross evidences of lower nephron nephrosis. The kidneys are usually swollen, the cortex is widened and pale, and the medulla is dusky and congested.

The prognosis is very grave, a mortality rate of 90 per cent having been found if renal failure and oliguria develop. Death usually comes quickly, but if the patient survives the first week, life expectancy is increased. Strauss[14] believes that the cause of death is either pulmonary edema or potassium intoxication.

If the patient survives the first week or ten days, recovery may be anticipated, and is usually sudden and dramatic. Copious diuresis occurs, azotemia declines and renal function rapidly returns to normal. As far as is known there is no residual renal damage.

The treatment of the lower nephron syndrome, though certainly far from satisfactory as evidenced by a mortality rate of 50 per cent to 75 per cent, has been delineated to such a degree that a synopsis of suggested methods will prove helpful.

In cases of crush injury Bywaters advises that fluids and alkali in the form of soda bicarbonate be given freely by mouth even before the patient is moved or before the pressure on the limb is relieved. He further warns against warming of injured limbs, as vasodilatation produced by heat may counteract the body's protective vasoconstriction and thus precipitate shock.

The prevention of shock is obviously the primary aim in treatment, and to this end whole blood, plasma or saline should be administered plentifully and promptly. The phase of shock usually lasts only a few hours and is followed by the onset of renal failure as evidenced by oliguria. The phase of oliguria requires close attention to fluid loss and electrolyte alterations, and most writers suggest a fluid intake of 750 c.c. to 2,000 c.c. In the presence of edema, fluid is further restricted. Strauss advises 750 c.c. of 15 per cent glucose in distilled water daily by vein, nothing being given by mouth. If vomiting occurs, he recommends that the volume of vomitus be measured and an equal amount of normal saline added to the intravenous infusion. In the presence of acidosis he suggests the use of 1 liter of 1/6 M. sodium lactate solution to replace the daily injection of distilled water.

A low blood sodium level may be raised by the intravenous administration of 5 per cent sodium chloride. If excessive potassium appears in the blood, intrajejunal perfusion through a Miller-Abbott tube with hypertonic glucose may be helpful. Hypocalcemia, of course, calls for the addition of calcium to the daily infusion.

Some writers urge that sodium bicarbonate be given freely to produce an alkaline glomerular filtrate, but others point out that such therapy is ineffectual, in that the alkali obviously does not reach

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the glomeruli in the presence of cortical ischemia. Also some[15] urge that fluids be pressed to a minimum limit of 3 to 4 liters a day, but, again, in a state of oliguria or anuria, such therapy is futile, and only edema can result.

In the period of recovery, when diuresis begins, fluids should be given in amounts equal to the urine volume plus at least 1,000 c.c. with sodium chloride to replace the salt lost in the previous day's urine. Transfusions again may be now used to restore the blood hemoglobin and protein levels.

The "artificial kidney", transperitoneal lavage, and jejunal lavage have aroused much enthusiasm in recent years, and subsequent refinements and simplifications may make such techniques of value for general use. It may be well to temper our enthusiasm for such methods, however, by rereading Strauss' paper in which he cites the cases of those who have survived four or more weeks without renal function and then have experienced spontaneous diureses and complete recovery. Two other cases have also recently been reported by Fowler and Hunt.[16]

The recent sensational discoveries resulting from the clinical use of the new adrenocortical tropic hormone may point the way to a definitive treatment of the lower nephron syndrome. If the adrenal hormones are able to defend the organism from the assaults of its environment, perhaps these hormones may be able to prevent or diminish the shock and renal failure which characterizes this syndrome. For the present, however, our aim, following the treatment of the initial shock phase, should be to try to keep the patient alive for the first week, for then recovery usually follows.

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DISCUSSION

DR. FRANK POLE, Richmond: I should like to thank Dr. Pinckney for presenting this paper to us. In the past several years so much that has been confusing and contradictory has been said and written about lower nephron nephrosis that Dr. Pinckney has done us a real favor in clarifying and summarizing the best of modern thought on this subject.

As a urologist, I should like to emphasize a few points brought out by Dr. Pinckney's presentation.

First, the treatment of lower nephron nephrosis has been shown to be nonsurgical in the majority of instances. We have had the experience of seeing patients who have apparently been relieved of their anuria by renal decapsulation or some other surgical procedure; but, in restrospect, we wonder if these patients did not recover in spite of rather than because of this surgery. Operation has usually been undertaken as a last resort; and, as in Dr. Pinckney's case, this is about the time that diuresis will appear anyway if the patient is going to recover at all.

A second point that should be emphasized is that fluids should be given to these patients in very limited amounts, although it has been the tendency of most of us to attempt to stimulate diuresis by the administration of venoclyses in large amounts.

Third, and last, it should be pointed out that the laboratory plays an absolutely essential role in the management of lower nephron nephrosis. The maintaining of proper electrolyte and acid-base balance will be the deciding factor in many cases; and very frequent laboratory studies, especially blood chlorides and CO_2 combining power, must be carried out.

Again, I should like to thank Dr. Pinckney for giving us the opportunity to hear an excellent presentation of a very interesting and important subject.

DR. PAUL D. CAMP, Richmond: I enjoyed Dr. Pinckney's paper very much, and I primarily want to ask his opinion on something. We see, particularly after myocardial infarction, quite frequently the syndrome in which there is a marked drop in blood pressure, perhaps down to even 90 or 80, or sometimes below that; or we see, in patients who have had beforehand a quite high blood pressure, a relative drop, say of even 100 milligrams of mercury. In these patients there is oliguria and sometimes even anuria. They go on to uremia, and the mortality rate is, of course, alarmingly high there. I should like to know, if in his opinion, the cause of oliguria and anuria and renal failure is due to a shunting mechanism of any type or due to

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lower nephron nephrosis or is due to simple lack of filtration pressure in the glomeruli which is brought about by the general fall in the blood pressure. I am sure I do not know the exact cause, and I do not know whether it is known or not. We are, then, between the devil and the deep blue sea, as the saying goes, as to what to do about these patients because we certainly do not like to give them too much fluid, and yet there is a feeling, on the part of some at least, that perhaps plasma followed by saline will bring the blood pressure up in some of these cases, and if it could be proven that it is due definitely to a shunting mechanism, then I think our mode of treatment would be different.

I should just like to ask Dr. Pinckney to comment on that.

DR. PINCKNEY, closing the discussion: I am naturally, in my ignorance, unable to answer Dr. Camp's question. I wish I knew. I should suspect, though, and think that the type of shock that would occur following coronary-artery thrombosis is of the central type, producing failure throughout the entire kidney, and that the kidney participates in the collapse, rather than there being any local shunting mechanism. Dr. Homer Smith a month ago said that he believes Trueta's renal shunt has no significance in the human being. Also, we must remember that persons suffering coronary accidents do not have normal kidneys in the first place. I should say there is general circulatory collapse and probably no localized renal shunting mechanism.

Neurologist Says Epileptic is Pilloried Unjustly.

The epileptic is unfairly pilloried in public opinion and law, according to Dr. William G. Lennox, a Boston neurologist, writing in the June 9 J.A.M.A.

Dr. Lennox, who is assistant professor of neurology at the Harvard Medical School and with the Children's Medical Center in Boston, said advice regarding marriage and children in such cases must be individualized.

He stated "Assets that are transmissible, such as sound vital organs, good intellect, personality and social responsibility, may outweigh the liability of a tendency to seizures."

"On the basis of family histories, the hereditary factor in epilepsy appears to be approximately the same as that in diabetes, one-half that in obesity and one-eighth that in migraine, but no state has as yet forbidden migrainous persons to marry. Unjustified discrimination in this case is born of fear. The success of present day therapy of seizures should reduce both fear and discrimination."

He reported on a study of the history of epileptic seizures, if any, of 20,000 near relatives of 4,231 epileptic patients. The incidence in these was 3.2 per cent.

Dr. Lennox concluded that the child of the average epileptic parent has a 30 to 1 chance of remaining free of seizures, although in individual instances the chances might be 10 to 1 or 100 to 1.

"Conditions that minimize the likelihood of an epileptic child are a family history devoid of epilepsy or migraine for the spouse, as well as for the patient, and minimal abnormality of their electroencephalograms [electrical recording of brain waves]; some acquired condition that is at least partially responsible for seizures; late onset of the illness and a normal mental endowment."

He stressed that in viewing the prospective parents, when one is epileptic, the question to be answered is: "Are the transmissible traits of mind, heart and body of such quality that reproduction is justifiable or perhaps advisable?" On the answer depends whether patients should be discouraged or encouraged, he said.

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OBSERVATIONS ON THE ANTIHISTAMINE DRUGS*

Oscar Swineford, Jr., M.D.,†
Charlottesville, Virginia.

Antihistamine compounds are useful when used properly. One manufacturer sold over $6,000,000.00 worth in a recent year, when there were about a dozen competing compounds. A billion antihistamine pills is probably a conservative estimate of the 1949 sales. It is unlikely that a large percentage of these were used properly.

The primary purpose of this report is to outline the uses, limitations, side effects and complications of these compounds. In addition, there are comments on their history, mode of action, chemical variants, pharmacological properties, and on why they are not the hoped-for panacea for the allergic diseases.

Historical: In 1911 Dale and Laidlaw noted the similarities between the effects of histamine and anaphylaxis in the guinea pig. This started the search for drugs to neutralize or prevent the effects of histamine, in the hope that allergy symptoms could thus be prevented. The first effective ones were the sympathomimetics, epinephrin and ephedrine. Many effective variants of these have been developed. They tend to correct the damage done by histamine and the atopic type of reaction, by relaxing smooth muscle and by hastening the absorption of exudate. Arginine, cysteine, and histidine have some antihistamine effects but are too toxic for clinical use. The Xanthines relax the bronchospasm of asthma. Aminophyllin, probably by virtue of its ethylenediamine content, has antihistamine effects too.

Histaminase and histamine-azo-protein were, predictably, not effective clinically. The gullibility of the medical profession and of the medicine-taking public are exemplified by the meteoric histories of these two drugs.

The first real progress in the antihistamine field began with the Fourneau-Bovet phenolic ether compounds in 1933, followed by the ethylene-diamines of Staub in 1939. In 1942 Antergan, the first clinical compound, was described by Halpern. In 1945 Benadryl, then Pyribenzamine were introduced in this country by Loew and by Mayer. Since then many antihistamine compounds have been made available. Certain advantages are claimed for each. A partial list is in Table I.

Table I
Antihistamine Drugs
A Partial List

Antistine	Histadyl	Pyribenzamine
Benadryl	Hydryllin	Pyrrolazote
Chlorothen	Neoantergan	Tagathen
Chlor-Trimeton	Neohetramine	Thephorin
Decapryn	Perazil	Thenylene
Diatrin	Phenergan	Trimeton
Di-Paralene		

Uses—Table II: Antihistamine compounds are particularly effective in relatively mild atopic reactions in the nose and skin. For example, they usually

Table II
Antihistamine Drugs
Uses

Angio. Edema	Hay Fever
Atopic Dermatitis	Serum Reactions
Allergen Reactions	Urticaria

Food and Drug Atopic Reactions
Asthma, Occasionally
Contact Dermatitis
(Local Anesthetic and Sedative)

relieve or prevent mild perennial and seasonal hay fever, acute or chronic urticaria and angio-neurotic edema, and reactions to allergen injections. They may relieve, but not predictably: mild atopic dermatitis, contact dermatitis, serum reactions, gastrointestinal allergy, atopic reactions to foods or drugs such as headache from chocolate or urticaria from aspirin. They are only occasionally helpful in asthma. They help asthma more frequently in children than in adults. They rarely relieve severe allergic symptoms.

The short duration of the wave of interest in the treatment of the common cold was predictable. They may relieve some of the symptoms but they have no curative effects.

The administration of antihistamine drugs is quite simple. They are effective by mouth, three to six times daily, or as needed. Doses range from four to

*Read before the annual meeting of the Medical Society of Virginia, at Roanoke, October 8-11, 1950.

†From the Allergy-Arthritis Division, Department of Internal Medicine, University of Virginia Medical School, Charlottesville, Va.

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one hundred mg. in tablet, capsule, or palatable liquid. Parenteral administration has been recommended. The reason is not clear. Local applications as wet dressings or salves may relieve itching in various forms of eczema, in conjunctivitis and in rhinitis. There is a strong tendency for these drugs to sensitize when used locally on the skin[7] or in the eye.[8] This is a partial contraindication to their topical use. The selection of an antihistamine to use will be discussed later. Aerosols have been recommended for asthma and hay fever. This is rarely the treatment of choice. Their tendency to dry the mucosae may be considered a contraindication in asthma.

TABLE III
ANTIHISTAMINE DRUGS
SIDE REACTIONS

Drowsiness	Nervous	Nausea
Dizziness	Tinnitus	Diarrhea
Dry Mouth	Weakness	Abdominal Pain
Headache	Fatigue	Tachycardia

Side reactions are common (Table III). Because of them antihistamines should be used with caution. The first dose should be taken when the patient can go to bed if untoward symptoms occur. If the first dose or two have no undesirable side effects, it is unlikely that subsequent doses will. If this precaution is taken, a minimum of automobile accidents, false arrests for drunkenness, unwise business deals, etc., can be blamed on antihistamines. This precaution will not prevent the occasional complications which, however, seem no more frequent than from aspirin, antibiotics, and other useful drugs.

Complications (Table IV) are infrequent, fortunately. More complications can be expected, however, with continued wholesale use. The incidence of sensitization phenomena may become appreciable

TABLE IV
ANTIHISTAMINE DRUGS
COMPLICATIONS

Acute Psychoses	Convulsions	Sensitization
Agranulocytosis	Hemolytic Anemia	Periarteritis
Respiratory and Circulatory Depression		

in seasonal hay fever and asthma or other recurrent allergic phenomena. Sensitization occurs typically when drugs are used periodically. The other complications are due to overdosage, or to individual idiosyncrasies which cannot be anticipated.

The mode of action of these drugs is interesting. Their antiallergic effects are due largely to their ability to block a histamine-cell combination or to make it an inert one. How this is accomplished is not known. Their effects are not limited to the prevention of histamine reactions. The other effects of the so-called antihistamine compounds are important but are less well-known and are not widely utilized. For example: They may be good sedatives. Most of them are potent local anesthetics. They have definite anti-cholinergic effects. They may potentiate epinephrin by neutralizing histamine. Still less well appreciated are their quinidine-like, anti-hyaluronidase and spasmolytic effects.

These effects are summarized in Table V.

TABLE V
ANTIHISTAMINE DRUGS
MODE OF ACTION

Block Histamine - Cell Combination	
Sedation	Local Anesthesia
Anticholinergic	Spasmolytic
Adrenalin-Sparing	Anti-Hyaluronidase
Quinidine-Like	

The limitations of the antihistamine drugs (Table VI) should be known by every physician who prescribes them. They *rarely relieve severe* hay fever,

TABLE VI
ANTIHISTAMINE DRUGS
LIMITATIONS

Asthma	Arthus Allergy
Atopy—Severe	Contact Allergy
Complications	Tuberculin Allergy
Side Effects	Periarteritis, etc.

urticaria, angioneurotic edema, reactions to serum and allergen injections, or even mild atopic dermatitis and asthma. This is particularly disappointing since these are the cardinal indications for their use and since histamine is supposed to play such an important role in these conditions. The striking failure in asthma has not been explained. When it is explained it may yield useful information about the pathogenesis of asthma.

Troublesome side-effects and the complications are obvious limitations. Equally obvious but less widely appreciated are their complete lack of effectiveness in the tuberculin, periarteritis, and Arthus' types of allergy. In these manifestations of allergy the role of histamine has not been demonstrated, nor strongly postulated. Antihistamines are therefore not in-

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dicated. The relief of itching obtained by oral and topical antihistamines in contact dermatitis may be explained by their local anesthetic and sedative actions and not by their antihistamine effects. There is evidence, incomplete however, that histamine causes some of the skin injury in contact dermatitis.

Pharmacology (Figure I). Histamine is blocked from receptor positions in the tissue cells by $N^{CH_3}_{CH_3}$. Most of these compounds have in their composition, or decomposition, this general formula: $R—CH_2\ CH_2\ N^{CH_3}_{CH_3}$, where R is any large slightly basic radical representing about 75 per cent of the molecular weight. Benzene, Pyridine, Pyrimidine, Thenyl and polycyclic rings in various combinations are the usual components of R. These rings are attached to the common di-methyl amino-ethyl chain through O, N, or CH_2 radicals to modify side effects, toxicity, and antihistamine activity (Figure I). On this basis, Seyler[6] lists four groups of compounds: (1) Benadryl, Decapryn and Hydryllin, linked to R through the O atom. (2) Pyribenzamine, Neo-antergan and Neohetramine, linked to R through the N atom. (3) Diatrine, Histadyl, Thenylene and Chlorathen, also linked to R through the N atom, are grouped because they have the Thenyl ring. (4) A miscellaneous group, such as Trimeton, Antistine and Thephorin differ from the other groups and from each other. Feinberg[3] uses similar groupings.

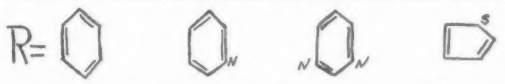
FIGURE I
ANTIHISTAMINE COMPOUNDS
SOME BASIC PHARMACOLOGICAL PRINCIPLES
(Modified from Seyler[6])

Antihistamine Activity	Drowsiness	Toxicity	Ring Compound Linkage and Variant		
2+	3+	3+	R - O - CH_2	CH_2	$N^{CH_3}_{CH_3}$ Blocks Histamine Tissue Reaction
3+	2+	2+	R - N - CH_2	CH_2	$N^{CH_3}_{CH_3}$
1+	1+	1+	R - CH_2 -	CH_2 CH_2	$N^{CH_3}_{CH_3}$ Side chain common to many Antihistamines

O—Linkage increases drowsiness and toxicity more than antihistamine activity.
Examples: Benadryl, Decapryn, Hydryllin.

N—Linkage increases antihistamine activity more than drowsiness and toxicity.
Examples: Pyribenzamine, Neo-Antergan, Neohetramine.

CH_2—Linkage decreases antihistamine activity and drowsiness and toxicity.
Examples: Trimeton, Chlortrimeton.



BENZENE, PYRIDINE, PYRIMIDINE, THENYL,
and various polycyclic ring compounds in arbitrary combinations designed to modify side effects and antihistamine activity.

There are many other pharmacological properties of the antihistamines, which have limited clinical application. The better known effects, not yet mentioned, are:

1. They neutralize the following effects of histamine: (a) Aerosol bronchoconstriction, (b) Spasm of guinea pig ileum, (c) Whealing of the skin, (d) Depressor action on arterial blood pressure, (e) Oxytocic action, (f) Increase in capillary permeability.
2. They protect against: (a) Active and passive anaphylaxis, (b) Specific contraction of the uterus of sensitized guinea pigs, (c) Experimental anaphylactic uveitis, (d) Experimental allergic nephritis. Large doses are required for most of these effects.
3. They prevent experimental orthostatic albuminuria in rabbits. 4. They may produce hyperglycemia. 5. They may inhibit oxidation of glucose and pyruvates in brain homogenates.
6. They inhibit, partially, the Schwartzmann phenomenon and the lesions of experimental chemical contact dermatitis.

They *do not* prevent: (a) Histamine production of gastric HCl. (b) Experimental meningoencephalitis, (c) Reversed anaphylactic reactions of the guinea pig to anti-guinea-pig-rabbit serum, (d) Arthus and tuberculin reactions.

Selection of an antihistamine drug for the individual patient. Fortunately, many patients tolerate and are relieved by the first one they try. If the first one does not relieve or is not well tolerated, the proper drug for that patient must be determined by trial and error. This process can be shortened by pre-

liminary trials of a key drug from each of Seyler's[6] or Feinberg's[8] groups. The final selection is made from the group indicated by the most effective least objectionable key drug. Remember that sedation is desirable as often at night as it is undesirable during the day. One drug at night and another for day use is common practice. These structural relationships are therefore of practical clinical significance.

Antihistamine compounds are not the long-hoped for panaceas for the allergic diseases. There are differences as well as likenesses between histamine effects and anaphylaxis, the animal reaction most comparable to human atopy. Histamine or its counterpart plays an important role in anaphylaxis, but so do other substances. The role of histamine has been studied widely. The roles of the other substances have not. For example, there is good evidence that hyaluronidase release, proteolytic enzyme activation, heparin release, lysocithin, potassium, cholinergic stimulation, and a slow-reacting substance (SRS) are concerned in anaphylaxis. So far, the antihistamines have exhibited protecting effects against histamine, acetylcholine, and hyaluronidase. It seems reasonable to suggest that solution of the problems of anaphylaxis and atopy is more apt to come from study of these, and of other unidentified substances, than from continued study of histamine alone.

Conclusions: The antihistamine drugs are useful. Their effects are purely symptomatic. They have no curative value. Their chief usefulness is in prevention and relief of some mild atopic symptoms. They do not, as a rule, control severe symptoms. Their sedative and local anesthetic effects can be utilized in selected problems. They seem to be little more effective than the sympathomimetic drugs, ephedrine and epinephrin, which have relieved symptoms of allergy for years. Unpleasant side effects are, perhaps, better tolerated than those caused by ephedrine and epinephrin, although they have not been compared adequately. They can be used in conjunction with the sympathomimetic drugs.

The antihistamine drugs are useful supplements to specific therapy. They should not be substituted for specific therapy except, perhaps, in the occasional case in which they afford complete objective and subjective relief.

Other properties than the antihistaminic ones need more study, especially the sedative, local anesthetic, spasmolytic, anti-cholinergic, epinephrin-sparing, quinidine-like and anti-hyaluronidase effects.

The antihistamine compounds do not, and can not be expected to, serve a significant purpose in the tuberculin and Arthus types of allergic reaction. Nor can they be expected to interrupt the genesis of the slowly developing allergic responses represented by periarteritis, rheumatic fever, glomerulo nephritis, and other diseases of hypersensitivity in which histamine has not been demonstrated to play a role. They have limited usefulness in contact dermatitis.

Physician and patient should be aware of the problems which may arise from the frequent side-effects and the infrequent complications.

The few references below contain a comprehensive bibliography of the whole subject of the antihistamine compounds.

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DISCUSSION

DR. WYNDHAM B. BLANTON, Richmond: Dr. Swineford has given us one of his usual clear and informative talks. I find in it nothing with which to take issue. There are, however, several points I should like to emphasize.

The first is the tremendous exploitation of these preparations. There are now 37 pharmaceutical firms which have taken 19 antihistamines, created 102 trade names, and distributed them in 123 different forms, deriving

from the procedure what has been conservatively calculated as an annual return of 100 million dollars.

Second, when the Food and Drug Administration allowed these drugs to be sold over the counter it did the American people an incalculable disservice. It put a very potent, toxic, sometime lethal drug in the hands of ignorant people.

From a bibliography which now numbers by my last count 587 articles, I gather that the antihistamines have been employed in about fifty different disease entities, in some with astonishingly good effect, such as hay fever, urticaria and other itching dermatoses, motion sickness, serum sickness, drug eruptions, etc. But in many other diseases they have been employed with questionable justification.

The treatment of the common cold with the antihistaminics is an example in point. There have been *now* enough reports of large-scale studies of patients given these drugs in the prevention and early treatment of colds to state *categorically* that what benefit may be derived is slight and probably no greater than that which can be achieved by other well-known drugs that are locally sedative, astringent, or analgesic.

An industry with which I am familiar experimented last year with Chlor-trimeton in combination with phenacetin in a large, fairly-well controlled series of cases. There were 8,000 employees who took, without knowing which, Chlor-trimeton and two placebos. The group that took a tablet of inert material reported in 53 per cent of the cases satisfactory results. The group that took Chlor-trimeton reported satisfactory results in 73 per cent of the cases. Now, I think that the difference between 53 per cent and 73 per cent is negligible in the light of the high incidence of reported improvement when given a fake capsule.

The weaknesses of this sort of experiment are evident. They consist in part in a reliance upon the patient's statement rather than upon objective evidence and in the failure to rule out the large number of cases of vasomotor rhinitis and instances of temporary nasal discomfort so frequently associated with fatigue, exposure to cold, or other irritating factors, which conditions closely simulate the common cold.

One series of experiments seems to me to settle the matter. Feller and his associates made direct virus inoculations from a patient with a cold into the nares of 49 volunteers. Sixty per cent took. Beginning just before the inoculation and continuing every four hours as long as there were symptoms, one-third of these recipients were given a recognized antihistamine, one-third another antihistamine, and one-third placebo tablets. Result: The percentage of colds, their duration, and their severity in each group were the same.

Finally, these drugs kill people. There are, it is true, only eight cases of death so far reported in the literature, most of which have occurred in children who have taken in large quantity cold cures bought over the counter and left around by careless parents. On the other hand, in Chicago a little while ago a man under the narcotizing influence of an antihistamine ran his automobile into another, killing two persons. How many industrial and automobile accidents can be attributed to the antihistamines has never been determined, but no one doubts the risk of their indiscriminate administration and the necessity for having the law changed.

DR. SWINEFORD, closing the discussion: I appreciate Dr. Blanton's excellent discussion. I do not see that there is anything to add to it. I should like to second all his warnings about the indiscriminate use of these potentially dangerous drugs.

Floral Eponym
Vallisneria

ANTONIO VALLISNERI (1661-1730)

Vallisneria or eel-grass is sometimes called tape-grass or wild celery. It is a favorite food of wild duck.

Vallisneri was a well known Italian naturalist, who was born in the city of Modène. He was professor of natural history at the University of Padua. He experimented upon the life and generation of insects, and was an opponent of the doctrine of spontaneous generation.

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BENIGN LESIONS OF THE STOMACH#

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In the limited time available, an attempt will be made to cover the vast subject of benign lesions of the stomach. We cannot go into detail on the many facets we would like to discuss but shall try to pick certain subjects which would be of general interest. Our exhibit on the same subject is on display, and I am sure it will answer some of the questions which will occur to you at some time during the presentation.

We have examined several books on general medicine[1,2], gastroenterology[3,4] and radiology[5,6]. Every author appears to have a classification of his own and not one seems to be completely satisfactory. We have assembled one of our own, derived from several sources[7,8,9,10], and based on etiology. Like other tables, it has disadvantages. Lesions caused by one group of agents may differ from each other and more closely resemble those in another group. However, the advantage of this classification is that, were the etiology determined, more definitive treatment could be instituted.

The stomach is a muscular hollow tube lined internally by a mucosa which is thrown up into folds and contains glands capable of secreting an acid secretion, pepsin, and mucus. It is covered externally by the serosa. It is nourished by an ample blood supply and drained by lymphatic channels. It is innervated by nerves from the sympathetic and parasympathetic nervous systems. The stomach reacts to injury or noxious agents in one of several ways, depending upon the extent of the insult. It follows naturally that a given cause may produce a greater or lesser effect in different individuals and that different causes may exhibit the same type of lesion. The pathologic changes that can occur are manifested by inflammation, atrophy, hypertrophy, degeneration, fibrosis, ulceration and tumor formation, no matter what the cause.

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CLASSIFICATION OF BENIGN LESIONS OF THE STOMACH

I. Congenital.
Absence of the stomach; thoracic stomach; transposition of the stomach; ectopic stomach; double stomach; inversion of the stomach; hour glass stomach; cascade stomach; micro and megagastrica; stenosis of the pylorus; atresia of the pylorus or cardia; pylorospasm; diverticulae.

II. Inflammatory; due to a specific organism.
- A. Acute phlegmonous gastritis, due to pyogenic organisms.
- B. Chronic gastritis, due to tuberculosis, syphilis or actinomycosis.

III. Non-specific, non-inflammatory.
- A. Acute gastritis.
- B. Chronic hypertrophic gastritis.
  1. General.
  2. Local; redundancy of mucosa with prolapse into the pyloric canal or esophagus.
- C. Chronic atrophic gastritis.
- D. Benign hypertrophic pyloric stenosis.
- E. Granulomas.

IV. Physical Agents.
- A. Irritants
  1. Foods; alcohol, condiments, strong tea and coffee; nicotine.
  2. Drugs; salicylates, ammonium chloride, quinine, cinchophen, aspirin.
  3. Swallowing of pathogenic bacteria and toxins.
  4. Ingestion of coal dust and silicates.

B. Corrosives.
C. Irradiation.
D. Trauma.
E. Foreign bodies, bezoars, gall stones.
F. Post-operative stomach — following anastomotic operations and post vagotomy.

V. Peptic ulcer.

VI. Benign neoplasms.
Polyps; adenomas; leiomyomas; papilloma; lipoma; phantom tumor; osteoma; cysts; myxoma; hemangioma; fibroma; lymphadenoma; neurofibroma; aberrant pancreatic tissue.

VII. Manifestations of Systemic Disease.
A. Arteriosclerosis.
B. Pernicious anemia; sprue; cord degeneration; iron deficiency anemia.
C. Polyarteritis nodosa.
D. Telangiectasia.
E. Lead poisoning; other organic and inorganic poisons.
F. Sarcoidosis.
G. Allergy.
H. Avitaminosis.
I. Stomach in skin diseases.
J. Many acute infections, as typhoid fever, influenza, etc.
K. Uremia.

VIII. Neurogenic and Reflex.

IX. Miscellaneous.
Varices; hernia; acquired diverticulae; volvulus; intussusception; calcification; pressure from extrinsic tumors.

Since we are chiefly concerned with the roentgenographic diagnosis, we can limit our discussion to a few representative groups. It is understood that no true diagnosis can be made without a detailed history, physical examination, certain laboratory tests and, in many cases, a gastroscopic examination. However, we wish to show cases illustrating typical lesions produced by various diseases.

The congenital group of lesions is self-explanatory and needs no further comment. They are not too common and are not of great moment, with the possible exception of congenital pyloric stenosis in infants. Certain of these, in particular, stenosis and deformities of shape, may also be the result of inflammation, peptic ulcer, carcinoma, corrosives, and neurogenic dysfunction.

Schindler[11] has stated that the most common lesion of the stomach is a gastritis. Not only may it be caused by numerous agents, but it commonly occurs in conjunction with other lesions. William Beaumont[12], in 1833, described the results of a series of experiments on Alexis St. Martin who had a gastric fistula. His observations were made under direct vision and are classic in their description. He described the changes in the stomach following the ingestion of food, liquids and other substances. He noted the swelling and redness of the mucous membrane, along with the oozing of blood and increased mucus secretion. Wolf and Wolff[13], also under direct vision, described the effects in the stomach after emotional stimuli. All of these changes can be seen in acute gastritis. There are many varieties of gastritis and Schindler[11] has made a rather detailed classification. It is stated that the diagnosis of acute gastritis should not be made unless a gastroscopic examination has been performed. Superficial and atrophic gastritis may show no roentgenographic findings. Many of you can remember patients who complained of bleeding from the upper gastrointestinal tract who showed no abnormal findings on X-ray examination. They probably fall into this class.

The two most common lesions which have to be differentiated radiographically are peptic ulcer and malignancy.

Gastric ulcers usually occur on the lesser curvature of the stomach, proximal to the incisura angularis up to one inch or more distal to the cardia. The niche usually projects well beyond the contour of the stomach and is smooth in outline. The ulcer is usually small and there may be other ulcers, especially in the duodenum. Opposite the niche a deep spastic incisura is present. However, all of these findings may not be present, thus leading to difficulties in diagnosis. To further add to the difficulty is the disagreement between the pathologists and the radiologists as to where the pylorus ends and the duodenum begins. This is important, because carcinoma of the duodenum is extremely uncommon, while carcinoma of the pylorus and prepyloric region is fairly common. A similar roentgenographic appearance may be observed in arteriosclerosis, syphilis, tuberculosis of the stomach, polyarteritis nodosa, Hodgkin's disease, chronic hypertrophic gastritis, following the ingestion of corrosives, and carcinoma.

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CASE REPORTS

Case I. J. G. T. White male, age 76, developed a lump in the left side of the neck six weeks before admission to the hospital on July 29, 1948. A section was removed for biopsy. Microscopic examination revealed the tissue to be neoplastic, simulating lymphoepithelioma of the nasopharynx (of Schmincke), but the type was not too definite. X-ray of the chest, barium enema and G.I. series were all negative. He was transferred to Veterans Administration Hospital, Richmond, Virginia, on September 10, 1948. The mass in the neck was irradiated and promptly disappeared. He complained at times of a vague sort of indigestion which was controlled by an ulcer regime. G.I. series, on September 21, 1948, was reported as showing a defect on the greater curvature of the midportion of the duodenal bulb, consistent with duodenal ulcer or periduodenal adhesions. He was readmitted to the latter hospital on December 17, 1948, for further irradiation of the left supraclavicular and right submental regions. A basal cell carcinoma of the right cheek was removed at the same time. He was readmitted to this hospital on February 28, 1949, for terminal care. He died on March 7, 1949. At autopsy there was a deep ulcer 5 cm. in diameter involving the prepyloric portion of the stomach, along the greater curvature, which had penetrated all the coats but had not perforated. The duodenum showed no ulceration. Microscopic examination of the ulcer base revealed Hodgkin's disease. In addition, there was involvement of the liver, spleen, and lymph nodes. The diagnosis was confirmed by the Army Institute of Pathology.

Case II, J. C. White male, age 48, was admitted to this hospital on October 17, 1946, as a transfer from another hospital because of vomiting of large clots of blood. He gave a history of stomach trouble since 1942, consisting of indigestion that appeared one to two hours after meals which was relieved by milk or antacids. He was discharged from the Service in 1942 with a diagnosis of duodenal ulcer. G.I. series revealed deformity, irregularity and tenderness in the prepyloric region of the stomach. Gastric analysis showed 70° of total acid with 35° of free HCl. A subtotal gastrectomy was performed on November 13, 1946. The Surgeon found ulceration of the pylorus on the posterior surface extending down to the duodenum. There were several small glands along the border of the stomach and there was in-

Fig I. Case I. J.G.T. September 21, 1948. Hodgkin's Ulcer of the Stomach.

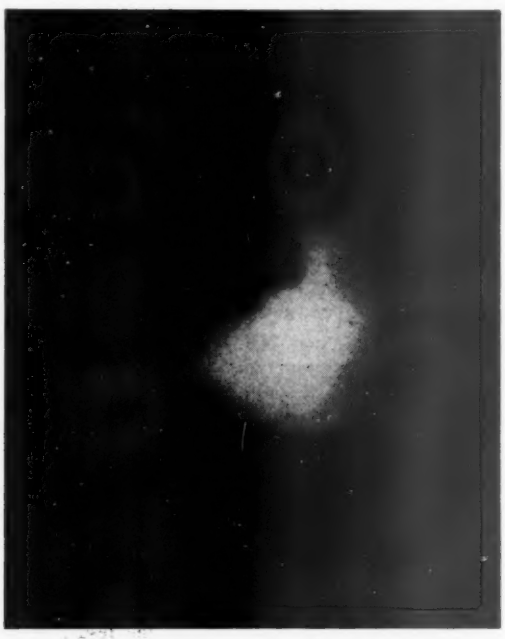

Fig. II. Case II. J.C. November 4, 1946. Polyarteritis Nodosa of the Stomach.

duration of the pancreas. Following operation he continued to run an irregular elevated temperature, developed weakness and numbness in his legs and unsteadiness in walking. He continued to lose weight and showed signs of avitaminosis. He developed an anemia and had a persistent leucocytosis. Repeat G.I. series showed no evidence of active disease. He died on May 13, 1947. At autopsy typical lesions of polyarteritis nodosa were found in the spleen and pancreas. Review of the slides of the resected stomach showed similar lesions. The Army Institute of Pathology agreed with this diagnosis.

Case III. M. H. Negro male, age 58, who had mild diabetes, CNS syphilis and transverse myelitis, reported to the laboratory for a blood sugar determination. The technician noticed that he was pale. He did a blood count and it showed a severe anemia with a hemoglobin of 25%. He was admitted to the hospital on December 8, 1947. He had been hospitalized here three months previously because of vomiting, dizziness, and unconsciousness. Following discharge from the hospital, he had several black stools. G.I. series, on January 6, 1948, showed an irregular filling defect involving the "pyloric inch of the stomach". The appearance was suggestive of a prepyloric ulcer. Gastric analysis after histamine showed a total acid of 44° with 22° of free HCl. The blood serology was negative. Repeat G.I. series, on February 17, 1948, was reported as essentially normal. He was readmitted to the hospital on November 22, 1948, because of hematemesis. G.I. series, on December 8, 1948, showed normal peristaltic activity. There was tenderness to palpation along the lesser curvature aspect of the stomach in the region of the pars media. In this area there appeared to be a small niche measuring 0.5 cm. in diameter. The appearance was that of an ulcer crater. There was no spasticity on the corresponding portion of the greater curvature. An exploratory laparotomy was performed on January 18, 1949. A tremendous carcinoma of the cardia was found. This had involved the posterior wall and had invaded the pancreas and liver. It definitely was inoperable; therefore, no further surgery was done. He died on January 19, 1949.

Case IV. C. M. W. Negro male, age 59, was admitted to the hospital on January 15, 1947, because of pain in the stomach occurring three-quarters to one hour after meals, and night pain. Relief was obtained by alkalis or milk. He had several hemor-

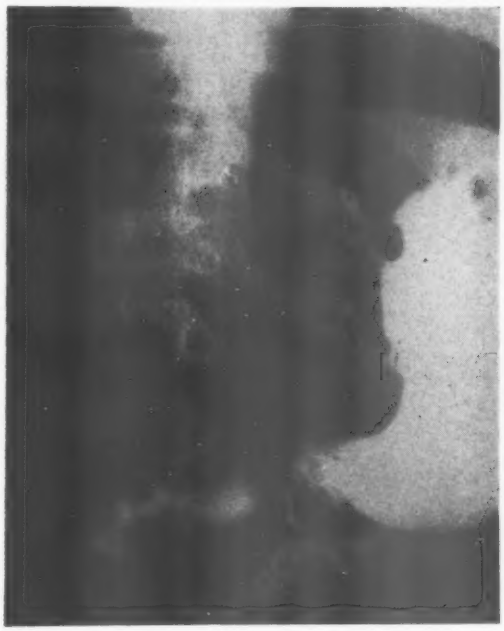

Fig. III. Case III. M.H. December 8, 1948. Carcinoma of the Cardia of the Stomach.

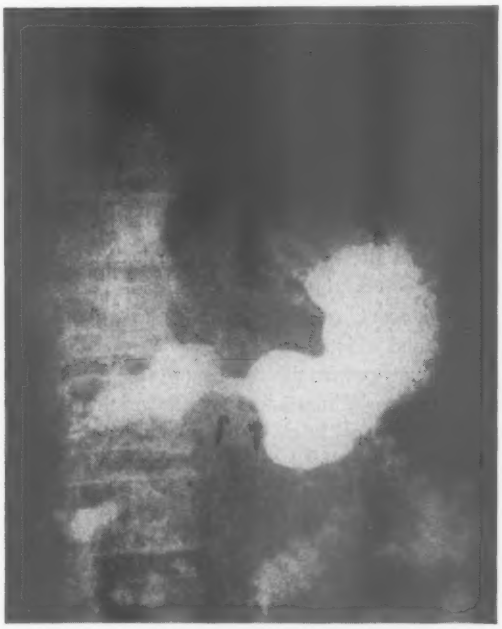

Fig. IV. Case IV. C.M.W. December 13, 1948. Hypertrophy of the Pylorus and a Fecalith in the Soft Tissues Between the Duodenum and the Head of the Pancreas.

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rhages. The onset of symptoms was in 1936. G.I. series, on January 14, 1947, prior to admission, showed a fairly large defect, measuring about 4.5 cms. in length, involving the pyloric portion of the stomach on its greater curvature side. No peristaltic waves could be seen passing through this area. This was interpreted as suggestive of a new growth in that area. He refused gastric analysis and surgery and was consequently discharged when his symptoms abated. He was readmitted to the hospital on October 3, 1947, because of vomiting and umbilical pain. Repeat G.I. series was done on October 20, 1947. There was tenderness involving the prepyloric portion of the stomach. This area was spastic and somewhat deformed. On the six hour film there was slight retention of the barium within the stomach. This was interpreted as showing an active prepyloric ulcer. Gastric analysis revealed free HCl ½ hour after the giving of histamine. He again refused surgery. The G.I. series was repeated in December, 1948, and March, 1949, and the same findings were observed. He was readmitted to the hospital on January 13, 1950, because of epigastric pain and the vomiting of blood. G.I. series was done on January 7, 1950. This again showed no change from the previous ones. An exploratory laparotomy was done on January 31, 1950. There was an area of moderate thickening of the stomach wall anteriorly near the greater curvature in the prepyloric region. The stomach was opened in this area but no signs of ulceration or inflammation were found. No further surgery was done. About three weeks later he developed a massive cerebral hemorrhage, from which he died on February 21, 1950. Autopsy revealed the stomach at the area of incision to be considerably thickened (affecting the mucosa and muscularis). On microscopic examination no remarkable changes were found. However, there was a fecalith in the soft tissue between the duodenum and the head of the pancreas. This had apparently originated from a ruptured diverticulum of the colon many years ago. This had caused the defect as seen on the X-ray films. Diverticulitis of the colon was also present.

The roentgenographic appearance of carcinoma of the stomach may manifest itself as an ulcer (especially if large and on the greater curvature), infiltration (meniscus sign), constricting fibrotic defect (hour glass stomach), defects due to pyloric stenosis and diffuse fibrosis (leather bottle stomach). However, these various signs are not all present at the same time and the same appearance may be given by various benign lesions, such as phlegmonous gastritis, benign peptic ulcer, syphilis of the stomach, chronic hypertrophic gastritis, benign tumors, granulomas, and neurogenic dysfunction.

Case V. R. H. K. White male, age 54, was admitted to the hospital on November 19, 1948, complaining of severe periumbilical pain, most marked in the mid-afternoon, of gradually increasing severity for the past two years. G.I. series, on November 24, 1948, showed some decrease in peristalsis in the antral portion of the stomach. There was marked hypertrophy of the rugae in the antrum, with irregular deformity on the lesser curvature aspect of the stomach distal to the incisura. There also appeared to be a shelf-like defect on the greater curvature aspect of the stomach in the distal pyloric portion of the stomach. This area was tender to deep palpation. This was interpreted as suggestive of a malignant ulcer and hypertrophic gastritis. Two repeat G.I. series were made, the last one about two months after admission, and both showed no change. Gastric analysis showed a total acid of 97° while the free HCl was 83°. A subtotal gastrectomy was per-

Fig. V. Case V. R.H.K. December 13, 1948 Hypertrophic Gastritis.

formed on January 14, 1949. The surgeon found a mass in the antral portion of the stomach that had the consistency of redundant gastric mucosa. This had a tendency to herniate into the duodenum, the first portion of which was markedly enlarged. The herniation could be easily reduced back into the stomach. Examination of the specimen revealed prominent rugae which appeared to converge towards the midportion of the lesser curvature. There was no evidence of ulceration. On microscopic examination only a chronic gastritis was found.

Case VI. D. C. White male, age 53, was admitted to the hospital on September 28, 1947. He complained of symptoms of three weeks duration consisting of epigastric pain, fainting, tarry stools, weakness, anorexia and a 20 lb. weight loss. He vomited "coffee ground" material on one occasion. G.I. series, on October 6, 1947, showed a large smooth spherical defect measuring almost 4 cm. in diameter on the posterior wall of the stomach proximal to the incisura. The central portion of the lesion showed a crater measuring 1.5 cm. in diameter. No rugae could be seen running through this area. Gastric analysis showed 133° of total acid, while the free HCl was 90°. Exploratory laparotomy was done on October 28, 1947. A mass was found within the wall of the stomach on the anterior surface in the mid body near the lesser curvature. It could be easily displaced and a crater could be felt in it. The stomach was resected. The specimen showed a polyp-like mass 5 x 3½ cm., on the surface of which were two ulcerated areas. On microscopic examination this was shown to be a neurofibroma.

Case VII. W. H. B. Negro male, age 68, was admitted to the hospital on January 3, 1949. About six months prior to admission he began to develop cramping pain in the left part of the epigastrium radiating to the left flank and to the precordium. The pain was nearly constant and not affected by food or soda. He had lost considerable weight. However, he had had some trouble with his stomach for the past 35 years (abdominal pain, occurring one to two hours after eating and lasting for one or more hours). G.I. series on January 10, 1949, showed decrease in peristaltic activity of the stomach. There appeared to be marked narrowing of the antral portion of the stomach with some upward displacement of the proximal portion of the antrum. This portion of the stomach was somewhat rigid and tender to palpation. This was interpreted as consistent with

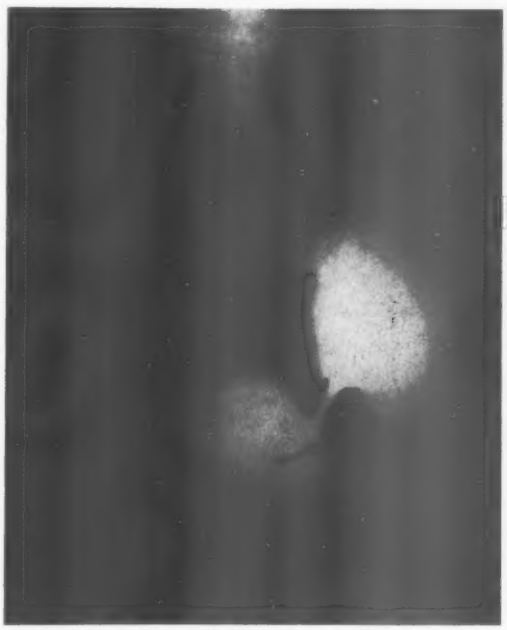

Fig. VI. Case VI. D.C. October 6, 1947. Neurofibroma of the Stomach.

Fig. VII. Case VII. W.H.B. January 10, 1949. Chronic Gastritis.

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a new growth. Gastric analysis showed a total acid of 18° with 4° of free HCl. A subtotal gastrectomy was performed on January 19, 1949. The surgeon found only prepyloric thickening of the stomach wall near the lesser curvature. The specimen showed normal rugae and no evidence of ulceration, past or present. On microscopic examination only a minimal amount of chronic gastritis could be found. He died one week after operation from pulmonary edema. At autopsy no signs of malignancy were found anywhere.

Case VIII. H. L. C. Negro male, age 29, was admitted to the hospital on March 29, 1949, stating that for the past five months he had gas and vomiting. He had lost 30 pounds in weight. He had had no pain. G.I. series, on April 6, 1949, showed decreased peristalsis. The antral portion of the stomach appeared to be elongated and greatly narrowed, and only a thin stream of barium could be seen passing through this portion of the stomach into the duodenum. There was marked tenderness to palpation over this area. On the 6 hour film the stomach showed complete retention of the barium. This was interpreted as suggestive of a new growth. Gastric analysis showed a total acid of 6° with no free HCl.

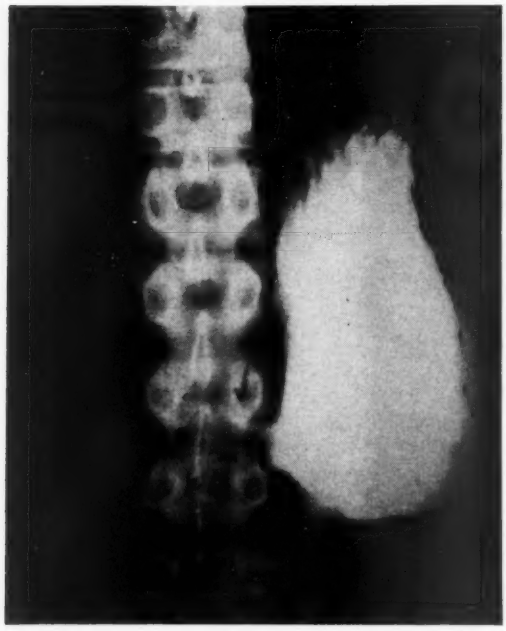

Fig VIII. Case VIII. H.L.C. April 6, 1949. Syphilis of the Stomach.

The Wassermann and Kahn were strongly positive. He had received 6,000,000 units of penicillin I.M. for syphilis in the Army six months previously. He received another 6,750,000 units of penicillin I.M. while in this hospital. Because his symptoms were unrelieved, an exploratory laparotomy was performed on May 5, 1949. There was cicatricial contracture and obstruction of the pylorus with thickening and induration of the prepyloric region for 1½ inches proximal to the pylorus. There was hypertrophy of the lymph nodes about the duodenum and the lesser curvature of the stomach. A subtotal gastrectomy was performed. The specimen showed the pylorus to be narrowed and there was a prepyloric ulcer 6 x 6 cm. completely encircling the stomach in this area and extending deeply into the muscularis. The microscopic findings were compatible with a syphilitic ulcer. The Army Institute of Pathology agreed with this diagnosis.

Benign neoplasms show nothing distinctive radiographically and may simulate either ulcer or malignancy.

Foreign bodies of all sorts have been found in the stomach. They chiefly occur in children and in the insane. If they are metallic they are readily visualized. Bezoars are composed of either animal, vegetable, or mineral products. On X-ray examination they may simulate a large gas bubble which is movable.

It would be obviously impossible in this short paper to describe the roentgenographic appearance of the stomach in all the diseases listed in the classification. We have tried to show that the X-ray appearance is not pathognomonic of any one lesion. Similar lesions may be produced by various agents. A rather large etiologic classification has been presented to show that the stomach is more frequently involved in disease states than we have been aware. This should stress the value of the history, physical examination, laboratory aids and gastroscopic examination in making the diagnosis.

SUMMARY

1. An etiologic classification of benign lesions of the stomach has been presented.
2. The presentation was limited to the roentgenographic appearance of lesions.
3. Cases were presented which showed typical

roentgenographic findings of ulcer or carcinoma, but which were due to a variety of causes.

4. This was explained on the basis of the physiopathologic response inherent in the tissues that make up the stomach.
5. It is important that we keep in mind that the stomach is so frequenly affected by disease and from so many different causes.
6. The roentgenographic appearance is suggestive but not pathognomonic of any one lesion. In fact, some lesions may not be visible radiographically.
7. It is urged that before a definite diagnosis of gastric disease is made on roentgenographic findings, an adequate history be taken, a good physical examination be performed, and the patient evaluated as a whole. Laboratory examinations of various sorts and gastroscopic examination should then be done to further clarify the diagnosis. Even then, the exact diagnosis may be in doubt to await the result of treatment and serial X-rays.

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DISCUSSION

DR. C. M. CARAVATI, Richmond: The interesting and unusual material presented demonstrates decisively that a conclusive *clinical* diagnosis of gastric lesions is not often possible. Barium studies and gastroscopy are the only means of localizing the pathology; but these often fail in determining the etiology, except when a representative biopsy can be secured through the operating gastroscope. The pathologist not infrequently cannot ascertain the exact diagnosis, as in *Case VIII* of the authors, where the microscopic findings were suggestive but not conclusive of lues.

The most common benign gastric disorders are gastritis and ulcer. Gastritis, only of the hypertrophic type, may be suggested by the radiographic appearance and confirmed by direct visualization with the gastroscope. The following cases are illustrative:

Case No. 1. History and X-ray suggested lymphosarcoma. At laparotomy and biopsy hypertrophic gastritis was demonstrated.

Case No. 2. Story of pyloric obstruction. X-ray showed constant prepyloric defect, thought to be cancer. Gastroscopy showed chronic hypertrophic gastritis, which was confirmed after surgery.

As the authors emphasize, benign peptic ulcers are most commonly found on the lesser curvature, proximal to the incisura angularis. In contrast, ulcerative defects in the prepyloric and apparently on the greater curvature are expected to be found malignant, though experience has shown that many of these lesions are essentially benign.

Case No. 1. Typical peptic ulcer on lesser curvature with healing under appropriate treatment.

Case No. 2. Prepyloric lesion healed and patient asymptomatic now for seven years.

Case No. 3. Extensive prepyloric, greater curvature ulcer in seventy year man thought to be cancer, but proven benign. Recovery. Well after four years.

Case No. 4. Marked antral defect in fifty-three-year-old male, thought to have been malignant but found to be benign.

The authors' experience and the above cases briefly outlined serve to emphasize that with our present armamentarium, including X-ray, gastroscopy and Papanicolaou stains, a positive pre-operative diagnosis of gastric pathology is seldom possible. By inference, Dr. Nushan and his co-workers stress the importance of the intelligent use of all available procedures, including surgery, to determine the etiology of a given lesion.

We are indebted to the authors for presenting to us many incidences of unusual gastric pathology and a new and practical classification of "Benign Lesions of the Stomach".

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THE DIAGNOSIS AND TREATMENT OF THE MIGRAINE SYNDROME AND RELATED VASCULAR HEADACHES*

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Headache as a complaint or symptom is encountered almost daily by practically all physicians regardless of their specialty. At one time or another almost 100 per cent of our population suffers from some type of this malady. During the recent war, studies of 10,000 army men between the ages of 18 and 36 revealed that 8 per cent suffered from "sick headaches".[1] Other investigators seem to substantiate this incidence.[2,3] It has been estimated that at least 12 million persons suffer from migraine in this country. No one knows how many additional patients suffer from related migraine variants.

It is generally thought at the present time that the migraine headache and its variants are intimately related in part to disturbances of the cerebral vascular system. Time and space will limit this paper to a discussion of the current ideas about the pathogenesis, diagnosis and treatment of these so called vascular headaches.

PATHOGENESIS—ORGANIC

Pain from within the head does not emanate directly from the parenchyma of the brain but from its supporting and vascular structures.[4] Wolff[1] and others have shown conclusively that migraine preheadache and headache phenomena represent derangements in cranial vascular function. Pre-headache phenomena which include scotoma, hemianopia, unilateral paresthesia, speech disorders and mood changes are apparently the result of cranial vasoconstriction. Evidence indicates that it is dilatation rather than contraction of arteries which causes vascular pain, although traction upon or displacement of certain cranial blood vessels may produce deep head pain.[1] The middle meningeal artery, arteries at the base of the brain, and some scalp arteries including the temporal arteries are equipped with afferent nerve fibers and conduct only those impulses resulting in pain. Headache, identical to migraine, has been reproduced by mechanical distention of the cranial arteries and relieved by inducing vasoconstriction.[1] Constriction and dilatation of certain cranial arteries, then, in susceptible people, can and do produce preheadache and headache phenomena.

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PATHOGENESIS—FUNCTIONAL

The vascular theory does not explain entirely the etiology of these complex headaches. Why they should occur in some and not in others is not clear, nor is the high incidence of headaches at the time of menstruation explainable. It has long been known, however, that persons suffering from vascular headaches usually have a similar personality pattern. As a rule these people are overly conscientious, meticulous and perfectionists. They are tense, striving individuals, and as they grow older and less efficient their failure to achieve perfection leads to deep seated frustration. They become intolerant of their failures in themselves and impatient and irritable with others. It seems that these people are pre-disposed and equipped with certain psychobiologic patterns which make them susceptible to pernicious emotional states. A constellation of disturbing influences swiftly develops into violent emotional patterns, which, in turn, precipitate the chain of reactions leading to severe head pain.

TYPES OF VASCULAR HEADACHES

Although closely related in origin both organically and functionally, vascular headaches can be divided into three specific clinical types; migraine, histamine cephalgia and tension headaches.

MIGRAINE

Migraine attacks are characterized by their periodicity and often by a family history of similar attacks. They usually are preceded by an aura which may be in many varied forms, which include scotoma, paresthesias, mood changes, diplopia or hemianopia, speech disorders, etc. These prodromal phenomena last from a few minutes to an hour, followed by a brief symptom-free phase, and are thought to be due to cranial vasoconstriction.

Then, however, the cranial vessels dilate, and the headache begins and becomes progressively worse.

It is usually unilateral and is an intense throbbing pain. Photophobia, mood changes, nausea and vomiting develop. The intensity of the pain may vary, and the attack may last for minutes, hours or days; and in the longer episodes the patient becomes markedly depressed, impulsive and hostile. Other bodily functions may be involved and to varying degrees. The attack eventually subsides and is often followed by a feeling of well being and frequently soreness in the neck muscles.

HISTAMINE CEPHALGIA

Closely related to migraine is another type of painful vascular disorders of the head described by Horton as "Histamine Cephalgia".[5] Pain is the outstanding complaint of these headaches. It is intense, constant and boring or burning in type. The pain is unilateral and usually conforms to ramifications of the external carotid artery involving the eye, temple, neck and face. At one time or another many people with this type of head pain contemplate suicide, so severe is the pain.

This type of headache is usually of short duration, lasting, as a rule, less than an hour. It frequently comes on at night after several hours of sleep. Its onset is very sudden and is generally helped by sitting or walking. Associated with this head pain is usually a sense of stuffiness of the nostril, watering of the eye and localized perspiration on the affected side. Nausea, vomiting and scotoma do not occur and there is no family history of headaches.

These headaches may be induced in the average patient who is subject to histamine cephalgia by the subcutaneous injection of 0.35 mgm. of histamine base. When this injection is given, usually an immediate generalized headache follows, but in about 30-40 minutes there is a severe unilateral histamine cephalgia which is indistinguishable from the spontaneous attack.

The exact mechanism of vasodilatation during histamine cephalgia is not known. Histamine, which is a powerful vasodilator agent, is probably liberated locally, but this is yet to be demonstrated. It is interesting, however, that the pain of migraine cannot be induced by giving histamine base hypodermically and that a large majority of patients with histamine cephalgia can be relieved by courses of histamine desensitization, as will be described later.

TENSION HEADACHES

Probably the largest group of vascular headaches are the tension headaches. These develop following or during periods of nervous tension. The pain, although not as severe as migraine or histamine cephalgia, is worse than the usual headache. It is generalized, but more intense in the occipital region. As a rule no prodromal symptoms precede the attack and, unlike migraine, there is no hereditary factor. As with most vascular headaches, however,

	MIGRAINE	HISTAMINE CEPHALGIA	TENSION HEADACHE
Heredity	Present in 60-70% cases	None	None
Prodromal Symptoms	Usually present	None	None
Duration	Minutes to days	Usually less than an hour	No pattern
Onset	Abrupt to gradual	Abrupt	No pattern
Time	During the day	Usually when asleep	End of day or weekend
Pain	Intense, throbbing unilateral or generalized	Severe, boring, burning unilateral	Generalized and less severe
Visual Disturbances	Usually present	Less frequent	Uncommon
Lacrimation, Nasal stuffiness	Unusual	Very common	Unusual
Nausea and Vomiting	Usually present	Rare	Unusual
Residual tenderness of head and neck	Common	Unusual	Common
Histamine test	Negative	Positive	Negative

CHART I. Salient Clinical Features in Differentiating Migraine, Histamine Cephalgia and Tension Headaches.

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there is usually an underlying emotional disturbance.

During periods of nervous tension, a state of hypertonicity exists. Eventually a period of hypotonicity follows which is due to "vascular fatigue" or to relief from the factors causing the nervous strain. Cranial vasodilatation occurs at this time and the pain-sensitive structures may be stimulated. The headache may be localized or generalized, depending on vessels involved.[4]

TREATMENT

In the treatment of the vascular headache, it is, of course, desirable to determine which type is represented. This is at times most difficult as there may be considerable overlapping. The classical features of each type are summarized in Chart I and are of help in differential diagnosis.

Treatment of the vascular headache should include both immediate symptomatic relief and measures which will prevent, if possible, the recurrence of the headache. So often this latter phase is entirely neglected.

The large variety of therapeutic measures used in the treatment of migraine indicates the lack of any specific treatment. In the past, surgical removal of various sympathetic ganglia, turbinates, and resection of certain of the cranial arteries and nerves have been tried with unsuccessful results.[6] Medical management including special diets, desensitization to various allergies, warm baths, vasodilators, massage, traction and such drugs as thiocyanate, calcium and potassium have not reduced the severity or frequency of migraine attacks. Recently Macy and Horton[7] have reported encouraging results with the use of intravenous histamine.

In treating the migraine attack the drug of choice is ergotamine tartrate parenterally. The efficiency of this drug depends upon its vasoconstricting ability which interrupts the pain producing mechanisms.[9] This drug may be given orally, rectally, or parenterally. Dihydroergotamine methane sulfonate (D.H.E.45) in 1 to 2 cc. doses intravenously is often more effective in very severe or prolonged migrainous attack or in the cases which cannot tolerate ergotamine tartrate orally.

The recent combination of 100 mgm. of caffeine with 1 mgm. of gynergan has proved to be the most effective oral or rectally given drug in treating the early migraine attack. The dosage of this drug is usually from 2 to 6 tablets, depending upon the severity of the attack and the individual response. The drug is contraindicated in patients with hypertension, pregnancy, liver disease, nephritis, or peripheral vascular disease and large doses should be avoided because of possible ergot poisoning. Hansel,[9] Ryan[10] and Horton,[11] and numerous others have reported enthusiastically on the effectiveness of this preparation in the treatment of migraine and other vascular headaches. Early observations in the Medical Clinic of the Outpatient Department of The Medical College of Virginia seem to confirm these reports and will be the subject of a later report.

Symptomatic relief for the attack of histamine cephalgia can usually be obtained by intravenous dihydroergotamine or by epinepherine 1:400,000 intravenously. Caffeine with gynergan, if taken early in the attack, is also very effective. Treatment of the individual attack does not usually constitute the problem that the headache of migraine does since these are of shorter duration. Prophylactic treatment with the use of histamine desensitization is of great importance in this type of headache. Horton[5] has advised that a solution of histamine diphosphate 0.275 mgm. per cc. be used. Treatment consists of subcutaneous injections given twice daily beginning with 0.1 cc. and increasing 0.05 cc. each dose. Occasionally an injection causes headache or flushing of the face and then the subsequent injection is reduced 50 per cent and the doses thereafter should be gradually increased as before. When 1.0 cc. per injection is reached, the time interval is gradually spread to 1.0 cc. every other day, and finally a maintenance dose of 1.0 cc. once or twice weekly is given indefinitely. By this time the great majority of these patients have received complete relief and of these it has been shown that in those who keep up the maintenance dose the likelihood of recurrence is markedly diminished.[7] Although with results probably not quite as effective as when twice daily injections are given, the author has obtained many excellent responses following the desensitization on a one injection a day routine.

Histamine desensitization may also be carried out intravenously. This is done by daily intravenous injections by the continuous drip method of a solution of 2.75 mgm. of histamine diphosphate in 250 cc. normal saline. The solution is given slowly and is discontinued as soon as symptoms such as flush-

ing, headache, or other reactions occur. Gradually the patient will be able to tolerate more and more of the solution until finally at the end of about two weeks the entire 2.75 mgm. can be given without symptoms. At this time, it is recommended that the patient be maintained on a 0.275 mgm. subcutaneous injection twice weekly.

In relieving the tension headaches, the underlying cause must be corrected. While this is being done efforts toward improving the basic vascular disturbance must be carried out. Relief from the headache often coincides with the vascular response. Ergotamine tartrate, dihydroergotamine or ergotamine with caffeine are the drugs of choice. Usually a mild sedative between attacks is beneficial.

More important than treating the immediate attack of head pain is the prevention of its recurrence. This may in part be influenced, as mentioned above, by histamine desensitization or by sedation, but in almost all of the cases of vascular headaches careful and skillful psychotherapy is indicated. The patient must be made to realize through convincing explanation that his headaches are the reactions or complaints of his body to the stress and strain of his own way of life. Ideally he should be taught to relax more, to decrease his day's activities by perhaps 25 per cent and to compete less with his fellow man. These people need emotional guidance, a sympathetic understanding and individual care from their physician. In the words of Sir William Osler in speaking of migraine, "the greatest folly of all is to treat all patients and every attack alike." The tablet or hypodermic should be regarded as only temporary aids in treating a chronic but often curable disease, and the incidence of attacks should be the true index of the success of treatment.

SUMMARY

The migraine syndrome, histamine cephalgia and tension headaches are currently thought to be due to derangements in cranial vascular function. Preheadache phenomena are probably due to cranial arterial vasoconstriction which occurs in hypertonic states. The period of hypotonicity which follows these states is accompanied by dilatation of one or more of the cranial vessels. Certain of these vessels have afferent pain fiber end organs and vasodilation causes stimulation of these fibers with resultant head pain. The usual person with vascular headaches has a common personality pattern. These people are overly conscientious, ambitious, meticulous and high strung.

The clinical features of these headaches are discussed in detail together with differential diagnosis. Therapeutic measures for the individual attack should include sedation and the proper use of ergotamine tartrate, dihydroergotamine or the preparation combining caffeine with ergotrate which can be taken orally. The prophylactic management of histamine cephalgia and migraine by histamine desensitization is discussed in detail.

Finally, the importance of psychotherapy in the prophylactic treatment of patients with vascular headache is stressed. It is felt that if adequate, patient and sympathetic attention is given these patients over a long period of time, together with sound emotional guidance and advice towards reorganizing their activities, that the frequency of the attacks of head pain can be markedly reduced.

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XUM

DISCUSSION

DR. GEORGE B. CRADDOCK, Lynchburg: Dr. Higgins has given us as clear and concise a discussion on vascular headache as is possible in the time allotted. He has described the type of patient in which these headaches are most likely to occur and the pathological physiology of the production of the headache. He has also outlined well a method of handling and treating these individuals. I shall try to elaborate a bit on a few points.

First, since the idea that migraine and epilepsy are related has been abandoned by the medical profession, the diagnosis of migraine can be made without stigma; and this diagnosis, with re-assurance of the absence of other organic diseases (as brain tumor), will be very helpful to many patients and in some, whose symptoms are not disabling enough to require further treatment, will be all that is necessary in handling them.

Another point is that, as Dr. Higgins has pointed out, most of these headaches become more numerous during periods of stress. However, it is more often after the stress has lessened, as during the first day or two of a vacation or during a week-end, that the headache will be most likely to occur. A person usually puts on his greatest "steam" to finish up his work and get his affairs in order before a vacation, and the first day or two of his vacation is when he is most likely to have the headache.

I disagree with one point in the time of the migraine headache. I have found it most likely to occur on awakening in the morning.

Hypertensive headache is a vascular headache about which something might be added. Wolff states that, other than the headache of hypertensive encephalopathy, other headaches in hypertensives resemble closely migraine headaches. He notes that many of these patients do not develop headaches until they develop sustained elevation in blood pressure. Others with headaches will become worse with the development of hypertension. Wolff feels that the added blood pressure increases the expansion of the branches of the external carotid, producing the increased expansile pulsations and hence the migraine-like headache. Wolff, however, points out that the level of the blood pressure at any one time is not related to the headache.

Dr. Higgins has given us an excellent description of the histamine headache. Wolff is of the opinion that this headache is simply a peculiar form of the migraine headache. An interesting case report by Alford and Whitehouse, however, of a soldier with both the histamine-sensitivity headache and a duodenal ulcer tends to refute this. When this soldier would have a histamine headache it was found that his gastric-acid curve was quite high and similar to his gastric-acid curve when histamine was administered. Between bouts of histamine headache the soldier's gastric-acid curve was quite low.

I have enjoyed Dr. Higgins' paper very much and appreciate the opportunity to discuss it.

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Clarke, Sir Charles Mansfield (1782-1857) Observations on those diseases of females which are attended by discharges. London: Longman, Hurst, Rees, Orme, and Brown, 1814. viii, 304 pp. 10 plates, 24.3 cm.

This is the only published work of the author. He describes an urethrocele as a thickening of the wall of the urethra and a dilatation of the blood vessels. The treatment consisted of scarification or the application of leeches. He introduced the term *carcinoma uteri.*

Sir Charles was at one time the leading practitioner of midwifery in London. He was educated at St. George's Hospital and the Hunterian School of Medicine. He was surgeon to Queen Charlotte's Lying-in Hospital. In 1830 he was appointed physician to Queen Adelaide, receiving a baronetcy in 1831. He was elected F.R.S. in 1825, and F.R.C.P. in 1836. He was admitted M.A. at Corpus Christi College, Cambridge in 1842 and became D.C.L. at Oxford in 1845.

PREGNANCY AND TUBERCULOSIS*

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The problem of pregnancy and tuberculosis has been and still remains one of the most controversial problems in medicine. For the most part, the available literature is a mass of confusion and the recorded statistics are unreliable because of the paucity of cases correctly studied. The viewpoints have varied from the teaching of Hippocrates who believed that "pregnancy is the best cure for consumption" to the opposite extreme: "if a virgin, no marriage; if married, no pregnancy; if pregnant, no confinement". In analyzing the voluminous literature on this subject, Jameson[1] in 1935 was unable to find a single report purporting to show the deleterious effect of pregnancy on pulmonary tuberculosis which was not open to criticism from the standpoint of incomplete data on the extent and type of lesion. He was impressed with the fact that many of the writers have not approached the problem with an open mind and have obviously been influenced by preconceived ideas based upon old precepts—admittedly a faulty attitude towards any scientific problem. The British Medical Association in 1936, in a special joint meeting of the internists, obstetricians and tuberculosis specialists, argued the subject concerning the effect of pregnancy on tuberculosis for a whole day and concluded that up to that time there were no studies available giving sufficient facts on a sufficient number of cases to justify an opinion[2]. Bridge[3] in May, 1947, stated, "In no branch of medicine is it possible to find so many diverse opinions, each one supported by charts, figures and percentages". Falls[4] in 1941 had this to say, "I am convinced that the gloomy picture that was painted in the textbooks of both medicine and obstetrics of a few decades ago, and the statistics that are still being quoted belong to an era in which both tuberculosis and obstetrics were badly mismanaged as compared with what we are prepared to do for these patients today. With the development of collapse therapy, adequate sanatorium management, local anesthesia and improved management of practically all the possible complications of pregnancy it would be strange indeed if we would have to accept the same viewpoint as our predecessors. Cooperative management based on a common understanding of the underlying pathology and therapeutic possibilities by both the internist and obstetrician is the solid foundation on which the intelligent management of these interesting cases should rest".

No attempt will be made to present a comprehensive review of the literature as excellent reviews can be found in several recent publications[1,5,6]. Johnson,[6] in 1948, reviewed 160 articles on the subject and presented a series of his own of 118 cases. He concluded that "therapeutic interruption of pregnancy should never be done except in extreme cases in which pregnancy without tuberculosis would be difficult, and in those in whom the other complications of pregnancy are serious". Fourteen of their 118 cases were worse after delivery but all fourteen were hopelessly sick with far advanced tuberculosis and had little, if any, chance for survival even if they had not been pregnant. Friedman and Garber[5] in another excellent summary stated that therapeutic abortion should rarely, if at all, be performed on patients with tuberculosis. Mariette[7] reported on two deaths which followed shortly after therapeutic abortions because of pulmonary tuberculosis and felt that aborting a tuberculous pregnant woman is more hazardous than allowing her to go to full term. McDowell[8] concluded from his observations that "therapeutic abortion is a dangerous and unwise procedure in patients with active disease". Williams[10] in his book on Obstetrics quotes the study of Bridgman and Norwood[11] who followed 134 cases for one year or longer and found that at the end of that time more deaths had occurred in those upon whom abortions had been induced than in those who were left alone.

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*Read before the annual meeting of the Medical Society of Virginia at Roanoke, October 8-11, 1950.

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Schaefer and Law[9] reported on 231 full term pregnancies which were divided into two groups; those, who were seen between 1920 and 1931 when all patients were treated by bedrest alone, and those who were treated between 1932 and 1938 when active collapse measures were employed. In the former group the mortality was the same for the pregnant and non-pregnant groups (thirty-two per cent and thirty-three per cent). Improved and unimproved case-comparisons were also about the same (33 per cent and 36 per cent). In the latter group the mortality rate for the pregnant group was 17 per cent, whereas for the non-pregnant group it was 26 per cent. They felt that pregnancy has no deleterious effect on tuberculosis and that the type and extent of the lesion plus the ante-natal and post-natal care were the important factors in the outcome of each patient. Forssner[12], of Stockholm, compared the results obtained in three hundred and forty-one women who had been delivered with three hundred and ninety-six other women who had not been pregnant. At the end of two years, it was found that the mortality was essentially identical in each group. He therefore believed that intervention is never indicated, and when practiced probably does more harm than good. Larson, Mariette and Litzenberg[13] reported on eighty-five women with tuberculosis who became pregnant, and concluded that "pregnancy has not been demonstrated to affect the tuberculous process adversely in the patients when they are properly treated for their tuberculosis". A most interesting report by Lyman in 1943[2] merits special consideration. Lyman was "trained in the school that held that all pregnancies complicating pulmonary tuberculosis should be terminated if seen before the third month, and also that women with any appreciable amount of tuberculosis should not marry". He practiced that viewpoint for many years in his position as Medical Director of the Gaylord Farms Sanatorium. However, after reviewing his statistics, he came to the conclusion that a woman with inactive disease who has shown no tendency to relapse after one year of active life could, if she took proper care of her health, "have a baby and, in due time, another one".

During the routine observation of our cases of tuberculosis who had become pregnant, we were impressed with the favorable outcome of the pulmonary lesions in spite of the fact that the majority of our cases refused hospitalization and chose, rather, to be followed in the clinic. In an attempt to clarify this problem and without bias as we had no definite viewpoint, we reviewed our cases of tuberculosis who had become pregnant. A total of one hundred and ten patients were studied who had gone through one hundred and twenty-eight pregnancies (Figure I).

FIGURE I

	Patients	Pregnancies
Pleurisy with effusion	1	1
Minimal	19	23
Moderately advanced	35	41
Far advanced	12	14
Pneumothorax	27	29
Reexpanded Pneumothorax	14	18
Thoracoplasty	2	2
TOTAL	110	128

No attempt was made to give a long-term follow-up as we were interested in ascertaining to our satisfaction, at least, what influence, if any, the pregnancy exerted on the tuberculous lesion and what course the lesion took in the two or three month post-partum

FIGURE II

	No. Preg.	No. Change	Improved	Worse
Pleurisy with effusion	1	--	1	-
Productive (fibrous)				
Minimal	17	17	-	-
Moderately advanced	22	21	-	1
Exudative (active)				
Minimal	6	--	6	-
Moderately advanced	19	4	11	4
Far advanced	14	3	-	11
Pneumothorax	29	29	-	-
Reexpanded Pneumothorax	18	18	-	-
Thoracoplasty	2	2	-	-
TOTAL	128	94	18	16

period. We do not feel that what might happen to a lesion two or three years after a pregnancy could possibly be blamed on that event.

PLEURISY WITH EFFUSION

There was one case of pleurisy with effusion which developed in a young colored female eight days prior to delivery. The fluid was positive on culture for acid-fast bacilli. Following an uneventful delivery she remained in the hospital for only two weeks. Following her release she was followed in the clinic and serial X-ray studies showed progressive and complete resolution of the pleuritic process.

MINIMAL LESIONS

In this group there were a total of twenty-three pregnancies in nineteen women. Seventeen of the lesions were of a productive, stationary character and in no instance was there any change in the lesion noted either during pregnancy or in the post-partum period. Six patients had active disease which showed progressive resolution and fibrotic changes during the pregnancy and post-partum. Although all of the active cases were advised to be hospitalized, only one consented to hospitalization and the remainder were followed in the clinic. No case showed any progression of the lesion.

MODERATELY ADVANCED

There were forty-one pregnancies in this group of thirty-five women with moderately advanced disease. Twenty-two of the pregnancies were in women with stationary lesions and none were hospitalized. Their lesions had been stationary for periods ranging from two months (two cases) to seven years prior to delivery, the majority for eighteen to forty-eight months. Twenty-one of these showed no change in their lesions either during the pregnancy or post-partum period, and one patient, whose lesion had been stationary for ten months, showed a very slight increase in the lesion one month post-partum which cleared on rest at home and remained stationary on subsequent follow-up. Nineteen of the pregnancies were in women with active disease, three of whom had cavitations. One of these patients gave birth to twins. Only three of this group were hospitalized, the remainder refusing hospitalization and were followed in the clinic. The three hospitalized patients made excellent recovery on bedrest alone. Eleven patients, even though not hospitalized, also showed progressive healing of their lesions following delivery. Four patients showed slight increase in their lesions within three months after delivery and five additional cases showed some progression in subsequent follow-up studies. In the three cases with cavitation two of them refused hospitalization. One showed no change in the lesion nineteen months post-partum and one showed a slight increase in the cavity eighteen months after delivery. One patient who was hospitalized signed a release two months after her baby was born and returned home. She was not seen again until the birth of her second baby fourteen months later at which time her X-ray showed a residual productive infiltrate with no evidence of any cavitation and she felt well and was asymptomatic.

FAR ADVANCED

In this group of fourteen pregnancies, six were in colored females with rapidly progressive caseous pneumonic lesions with bilateral cavitations. Three of the pregnancies were associated with no significant changes noted in their disease for as long as three years. The causes of the rapid decline in the others were terminal spontaneous pneumothorax (eighteen days post-partum), terminal hemoptysis (one day post-partum), exsanguinating vaginal hemorrhage (six days post-partum), broncho-pleural fistula (six weeks post-partum) and one patient died of complicating intestinal tuberculosis, her pulmonary lesion showing no change for seventeen months after delivery.

PNEUMOTHORAX

There were twenty-seven pneumothorax patients who had had an effective collapse at time of delivery. Two of these patients had a second child while still undergoing collapse therapy. Three of the patients had a maintenance oleothorax at the time of delivery; in two the mineral oil had been injected because of a tendency for the lung to reexpand and the third had had a replacement oleothorax because of an empyema. The duration of effective collapse ranged from three months to sixty months, most of the cases having had an effective collapse for twenty to thirty-six months. In no instance was there any change noted in the pulmonary lesion either during pregnancy or in the period after delivery. No patient experienced any discomfort because of their collapse during delivery. The collapse was continued in each case after delivery for variable periods of time.

REEXPANDED PNEUMOTHORAX

In this group, there were fourteen patients, four of whom had two pregnancies, making a total of eighteen deliveries. The duration of effective collapse ranged from twelve months to sixty months with a majority having their collapse for three years. The interval between complete reexpansion and delivery ranged from one month to fifty-seven months, with the majority having their babies twenty-four to thirty-six months after having been reexpanded. In no cases was there any alteration noted in the residual productive infiltrate seen on the X-rays after expansion. All of the cases had cavitation with a positive

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sputum prior to the induction of pneumothorax. One of the patients had had bilateral reexpanded lungs for thirty-six months prior to delivery and experienced no difficulty during labor and delivery.

THORACOPLASTY

There were two patients in this category. Both had had two-stage operations four years and six years prior to delivery respectively. In neither case was there noted any change in the collapsed lung.

It may be of interest to add here that in the entire group there were six patients who had had a permanent phrenicectomy. None of these experienced any difficulty during their pregnancy or during delivery.

DISCUSSION

In a brief analysis of our cases, we are impressed with the following facts: All of the cases with productive lesions, even though none were hospitalized after discharge from the maternity hospital, did well with the exception of one case whose lesions had shown no change for only ten months prior to delivery and she, after being placed on bedrest, showed prompt resolution of the slight increase of her disease. In the group of twenty-five active cases of minimal and moderately advanced disease, four showed some progression in the post-partum period, none of whom had consented to hospitalization. All returned to their homes to care for their children and household duties and, in spite of this, all four cases showed only a slight increase in the extent of the existing lesions. It is needless to say that any case of active tuberculosis when so diagnosed would certainly be urged to go on a strict bedrest regime and would be told that the prognosis would be poor if such treatment were not followed. It is, therefore, not surprising that several of these cases showed progression. The same, I am sure, would have happened, in any group of active male cases, where pregnancy would not have been a factor, should they have continued with their daily occupations. All of the cases undergoing collapse therapy and those whose lungs had reexpanded following a period of pneumothorax treatment, did well. Our data does not substantiate the frequently quoted statement that pregnancy aggravates a tuberculous lesion, for in no case did we see any increase of the lesion during a pregnancy. Nor do we feel that a well conducted delivery will cause an exacerbation of a tuberculous lesion. However, we do feel where there is an increase in the extent of the lesion in the period following delivery that this is the result of the greatly increased activities assumed by the patient. To be sure, where a patient with active disease refused hospitalization we always urge that they adopt and follow a bedrest regime at home but, from personal knowledge, we know that this was not done by all of our patients. All of them came from the lower-income levels and were forced, by reason of their poor economic status, to personally care for their homes and families and, in almost fifty per cent of the cases, there were other children at home that had to be cared for also.

As to what advice we would give a woman with tuberculosis regarding her becoming pregnant, we feel quite clear in the following statements. If she is receiving pneumothorax, we would advise her becoming pregnant after an interval of two years of effective collapse. That would allow for control of the lesion during the pregnancy and for approximately a year or more after delivery. If the lung has reexpanded or if it is an arrested case of tuberculosis, we would recommend a period of one to two years under ordinary activity to observe the lesions before permitting pregnancy. We would not of course, advocate pregnancy in a woman with an active lesion, even though we strongly are of the opinion that activity itself in a tuberculous lesion is not sufficient reason to interrupt a pregnancy already started[3]. That individual, because of the type of lesion which she has, would receive the same advice for her tuberculosis as would be given her as if she were not pregnant, such as bed rest, collapse therapy, etc., as indicated. On a number of occasions, we have seen patients for the first time in the course of a pregnancy with cavity lesions which we have collapsed satisfactorily during the pregnancy and have gotten an effective collapse by the time of their delivery. The use of the several therapeutic measures at our command, both in the form of collapse measures plus the more recent antibiotics, makes it even more possible to effectively treat our pregnant tuberculous women. Certainly, we do not regard therapeutic abortion as a substitute for effective anti-tuberculosis therapy.

It has been our observation that on occasions the physician's desire to abort may be influenced by the patient's desires. We agree with Bridge[3] when he states that "whether a child is wanted or not is hardly the immediate concern of the physician".

Conclusion

In a review of one hundred and twenty-eight pregnancies occurring in women with tuberculosis, we can find no evidence to feel that pregnancy or delivery exerts a deleterious effect on the tuberculous lesion. No patient in our series, excluding those with hopeless, far-advanced disease, who followed a bed-rest regime following delivery, showed any increase in their disease. Those patients with active disease who refused hospitalization after delivery and return to their homes to assume the cares and responsibilities of their home and children can be expected to show progressive changes, but this progression, we feel, is the result of the nature of their disease process with failure to take the prescribed treatment. Where a patient who is pregnant is seen with tuberculosis, all efforts should be directed towards the treatment of the pulmonary lesion and the pregnancy should not be interfered with. The use of abortions in pregnancy cannot be considered therapeutic, and certainly is no substitute for well-established therapy directed to their pulmonary lesion.

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203 Wainwright Building

Discussion

Dr. C. Lydon Harrell, Norfolk: I have listened to Dr. Kruger's paper with a great deal of interest and pleasure. He reported an interesting series of cases with excellent results, but he did not follow his cases long enough for final results. Like many others who write on the subject of tuberculosis in pregnancy, he discussed one side very thoroughly, but leaves the other side to the imagination.

One can readily see from Dr. Kruger's discussion that he is very much opposed to a therapeutic abortion under any circumstance. If one would take the trouble to search the literature he would find just as many students of tuberculosis in favor of therapeutic abortion in certain types of cases as opposed. The main trouble is that the subject has never been studied and followed sufficiently to reach a proper conclusion. The obstetrician is primarily interested in delivering a normal healthy baby and leave the mother's perineum in as good shape as possible. He sees the mother once after she leaves the hospital and reports good results. In all probability he is correct, as most mothers, even though they do have tuberculosis, do well while carrying the baby. He does not mention nor does he know what happens three or six months later—whether the mother breaks down or the child develops a tuberculous meningitis.

The internist probably never sees the case until it is too late to do anything except to advise the mother how to take care of herself while carrying the child and immediately following delivery; consequently, many go unrecognized until it is too late.

Every pregnant woman should have an x-ray picture made of her chest at the time of her first prenatal examination if any suspicious lesions are noticed, and she should be advised to consult an internist at once and follow his advice. The obstetrician and internist should work together. The cases should be followed for at least five years. One should know what happens to the mother, the child and the other children in the home. It is a well known fact that child rearing is harder on the mother than child bearing. By a cooperative follow-up study much information could be obtained which would be of great value in helping one to determine the line of treatment in other cases.

Dr. Kruger states that a number of his advanced open cases were permitted to return to their homes after leaving the lying-in hospital to care for their babies and other children in the home. This is contrary to all public health teaching and tuberculosis control program.

The question arises: If you had a pregnant woman with tuberculosis, what would you do? How would you decide whether to carry her to term or to abort her? First of all, the mother has the final say; without her consent nothing can be done. Assuming the mother is

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willing to take her physician's advise, one should approach the subject from three angles:

First, scientific or medical;
Second, from the standpoint of Public Health;
Third, socio-economical.

There are certain types of cases that many physicians will agree should be aborted if recognized before the fourth month of pregnancy:

1. The far advanced active case.
2. Those with cavity and in the exudative stage.
3. Hemorrhage cases.
4. Laryngeal or kidney complications.
5. Any case with a positive sputum.

On the other hand, minimal and moderately advanced cases may be taken safely through pregnancy if in competent hands, provided they are willing and financially able to stay on the cure, preferably in a sanatorium, until the time of delivery. After the delivery, the child should be taken from the mother and the mother returned to the sanatorium and remain until she is considered safe to live at home with children.

Certain cases with an open cavity may go through pregnancy provided the lung can be adequately collapsed and she stay on the cure. Pneumothorax once started on a pregnant woman should be continued at least two years after delivery.

In considering these cases, one should always think of the economic situation. It is not fair to the family nor to the community to tell a woman with tuberculosis that she can go through her pregnancy when you know she is not able to pay her way at a sanatorium or to employ help at home. She becomes a menace and a care on the community during the pregnancy and a double care after delivery.

As a public health measure, we should try to isolate every open case of tuberculosis and endeavor to have every child born and reared in a healthy environment.

I wish to thank Dr. Kruger for his excellent paper.

DR. DEAN B. COLE, Richmond: I had not expected to discuss this paper, but I think Dr. Kruger is over-optimistic. Certainly our results have been nothing like so good.

We have a number of patients who have gone through pregnancy following treatment for tuberculosis without relapse or other complications. Some of these were pneumothorax patients in whom collapse was continued during pregnancy and for a time following delivery. For the most part, these patients have gotten along well.

On the other hand, we have seen a number of tuberculous women who became pregnant against advice and who relapsed during pregnancy or shortly after delivery. We frequently see women whose first breakdown with tuberculosis was either during pregnancy or shortly after delivery and I am convinced pregnancy is not good for tuberculosis and may be quite harmful.

I think this problem, like so many others, must be individualized and handled accordingly.

DR. KRUGER, closing the discussion: I knew I was in for a lot of arguments if I presented my side of the story.

To go back at Dr. Cole first, no mention was made in the paper that I can recall where we say that pregnancy is good for tuberculosis. That has been stated, as a matter of fact, by some good observers. They felt that the tuberculosis seemed to do better during pregnancy. But that statement was never made in the paper.

Insofar as the statement regarding the occurrence of exacerbations of tuberculous lesions during pregnancy, I am sure that all men treating tuberculosis have occasionally seen exacerbations of lesions in individuals who were not pregnant and who were receiving excellent sanatorium therapy.

This subject is one that deserves further investigation and study. I am grateful for being allowed to present the subject to you and, even if it has only stimulated some of you to give the subject more thought and consideration, I will feel amply rewarded for my efforts.

Separate Babies from TB Mothers As Precaution Against Disease.

Babies born of tuberculous mothers who are removed from contact with the mother immediately after birth do not contract the disease, according to three pediatricians. Drs. Bret Ratner of New York, Alexander E. Rostler of Fall River, Mass., and Pierre S. Salgado of Port-Au-Prince, Haiti, writing in the *American Journal of Diseases of Children,* added that with proper diet these infants will grow and develop the same as infants born of healthy mothers.

According to the article, the incidence of prematurity in offspring of tuberculous mothers is exceedingly high, ranging from 23 to 64 per cent, depending on the severity of the disease in the mother.

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August 1, 1951

Case Reports

A 60 year old woman developed a sore throat for which she sought the advice of her family physician. He told her she had a foul ulcer in the left side of her pharynx and prescribed a gargle. She got some relief but it was temporary and after three months, her throat was more painful than ever. Her doctor told her that the ulcer was larger and referred her to an otolaryngologist. He obtained smears from the ulcer which were reported as negative for Vincent's organisms, cultures which showed colonies of streptococci and staphylococci and several Wasserman tests which were reported negative. He treated the bacterial infection with sulfa drugs and penicillin for ten days. Altogether, she was in this doctor's care for about two months. When there was no improvement, he referred the patient to another otolaryngologist. This physician found a necrotic ulcer involving the left anterior pillar, alveolar ridge, buccal mucosa, and hard and soft palates. The lateral surface of the left side of the tongue was indurated, and there was a hard, fixed mat of nodes beneath the angle of the left jaw. A biopsy from the margin of the ulcer was reported "epidermoid carcinoma, transitional cell type." After careful evaluation, the lesion was classified as inoperable. Therefore, palliative radiation was the only treatment given, and the prognosis is hopeless.

Comment: 1. There was a delay of five months between the time when this patient first saw a physician and the time when the diagnosis was reached. The delay was avoidable and the first two physicians involved must accept responsibility for it. If the diagnosis had been made when the patient first sought medical advice, she would probably have been eligible for an attempt at cure by surgery, one or more forms of radiation, or a combination of methods. Even if an attempt at cure had been made and later had proven unsuccessful, active comfortable life would surely have been prolonged.

2. The reason the first two physicians failed to make the correct diagnosis is the usual one — they did not think of cancer as a diagnostic possibility. They considered only various kinds of infection. Even most laymen now are aware of the fact that "a sore that does not heal" is reason to suspect cancer.

3. To practice adequate medicine, every physician must make the biopsy a part of his standard procedure. Failure to do so will inevitably lead the doctor into errors of disastrous consequence.

4. There is no longer any practical or economic reason to omit biopsies. Containers for mailing specimens may be obtained from most druggists and from the State Health Department. Through the State Health Department, physicians may obtain free pathological service for their needy patients from any of the certified pathologists in Virginia or from the Department of Pathology at both medical schools.

5. In the above case, the physicians were presented with a lesion readily accessible to biopsy. The first two physicians did not take a biopsy and therefore did not reach the correct diagnosis. The third physician recognized his opportunity, took a biopsy, and made the diagnosis which should have been made five months before.

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TREATMENT OF EPILEPSY WITH MESANTOIN* OR HYDANTAL**

A Study Of 35 Consecutive Patients With Grand Mal Seizures Treated At The Virginia State Penitentiary And In Private Practice With A Follow-Up Of 1 to 2 Years

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In approaching the complex difficulties of ferreting out specific causes responsible for the appearance of epilepsy and evaluation of therapeutic procedures, we must in all humbleness acknowledge our inadequacies individually. It is through the pooling of collective effort and study carried forward tenaciously but conservatively that we are able to achieve our goal. This principle is paramount in order to successfully surmount the obstacles of treatment.

There is a considerable aura of uncertainty prevailing regarding the determining of cause in epileptic seizures. The Jacksonian conception that a "discharging lesion" is the cause of epilepsy has led many responsible persons, including James Purdon Martin,[19] to believe that this provides insight into the explanation of many neurologic manifestations of a paroxysmal origin. Jackson states firmly that, "A convulsion is but a symptom," and implies that "there is an occasional, and excessive, and a disorderly discharge of nerve tissue on muscles." In accordance with this, we note that the most violent discharges take place at the cortical level. It is interesting to find that fibrillation in amyotrophic lateral sclerosis is apparently indicated as arising in the degenerating anterior horn cells.

In turning our attention to the thalamic syndrome, we find that Head and Holmes[15] in 1911 explained it as a resultant effect of released thalamic function precipitated by damage to higher centers normally controlling the thalamus. Classical evidence of the thalamic syndrome's presence is exemplified by explosive, hyper-acute and, at times, immediate pain in one side of the body.

The likelihood that the damaged thalamus may be a discharging lesion itself is conceded, as may also be the case in chorea, athetosis and spasmodic torticollis.

Nils Antoni[2] places emphasis on the advisability of broadening epileptic definition to encompass conditions not of necessity marked by "attacks of loss of consciousness with convulsions." We do not find tonic and clonic movements present in many epileptic forms and the degree of lost consciousness during seizures may vary greatly. "In addition to the purely visceral phenomena of grand mal, such as bladder contraction and pupil dilatation, there are seizures whose clinical characteristics are entirely visceral, called vasovagal seizures by Gower."[11] In studying the relation of auras to seizures we find that "auras are not necessarily sensory but may be characterized by motor phenomena, such as running a short distance, intellectual phenomena such as dreamy states, or emotional phenomena." Antoni[2] further concludes in reporting on uncinate fits that the only epileptic phenomena which definitely indicate the presence of abnormal function of the uncinate gyrus of the anteromodial part of the temporal lobe are olfactory and gustatory auras. The field of research regarding precipitating causes of these abnormal discharges has yet to be explored.

In reviewing the history of the development of drugs and treatment designed to combat epilepsy, it is worthy of note that there are three significant categories and periods. The first period began about 1853 with the inauguration of drug therapy in the category of bromides. Encouragement was gained, for this medication did indeed reduce the incidence of epileptic attacks, but regrettably with its use the *bete noire* of unwelcome side effects reared its ugly head in the form of bromism, a condition which, of necessity, should not be minimized.

The second period, which saw the appearance and quick consolidation of phenobarbital as a therapeutic agent offering bright hope to the unfortunate victims of epilepsy, began in 1912. We take occasion to note at this point that the merit and efficacy of this drug overshadowed bromides in the opinion of no inconsiderable number of workers. And in 1937 came the third period which marked the initiation of the drug diphenylhydantoin sodium (Dilantin), an

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achievement of appreciable magnitude, and for whose introduction Merritt and Putnam[21] should be duly commended.

While by no means being a perfect anti-convulsant drug, it is significant to note from a collective analysis of authoritative papers published, that it was far superior to any previous medicants in reducing epileptic seizures. For other studies on the use of this drug, one is referred to the reports of Merritt,[21] and Putnam,[22] Finkleman and Arieff,[10] and Lennox.[17]

Through further investigation of diphenylhydantoin sodium's potentialities, it was noted at times that undesirable side effects appeared with its use, such as gastric disturbance, interruption of muscular coor-

"SEIZURES"

Date: _____

Number: _____ Name: _____ Race: _____

When did you have your first seizure? _____

Can you remember any of the happenings before a seizure? _____

Did you bite your tongue? _____ Did you wet or mess up your bed? _____

How old were you when you had your first attack? _____

Frequency of attacks? _____

How long do the seizures usually last? _____

How long have you been taking treatment? _____

Type of treatment: Mesantoin: _____ Dilantin sodium: _____

Phenobarbital: _____ Other: _____ Dosage: _____

Results of treatment? _____

What are your feelings following an attack? _____

Do any members of your immediate family have epilepsy? _____

Do you know of any of your relatives who are afflicted with epilepsy? _____

Have you ever had a serious head injury? _____

If so, were you unconscious and how long unconscious? _____

Have you ever had any other serious injury? _____

Have you ever had a serious illness? _____

Do you have frequent headaches? _____ Dizziness? _____ Blind staggers? _____

Pressure sensations in head? _____ Fainting spells? _____

Loss of orientation and knowledge of whereabouts? _____

Use of alcohol: None: _____ Moderate: _____ Excessive: _____ Sprees, how often? _____

Do you use dope or narcotics in any form? _____

Do you get angry or depressed easily? _____

Type of epilepsy: Grand Mal: _____ Petit Mal: _____ Other: _____

Recession of attacks: Complete: _____ Mild: _____ Moderate: _____ None: _____

General physical condition: Good: _____ Fair: _____ Poor: _____

Do you feel that the attacks are milder or worse since treatment with Mesantoin? _____

_____

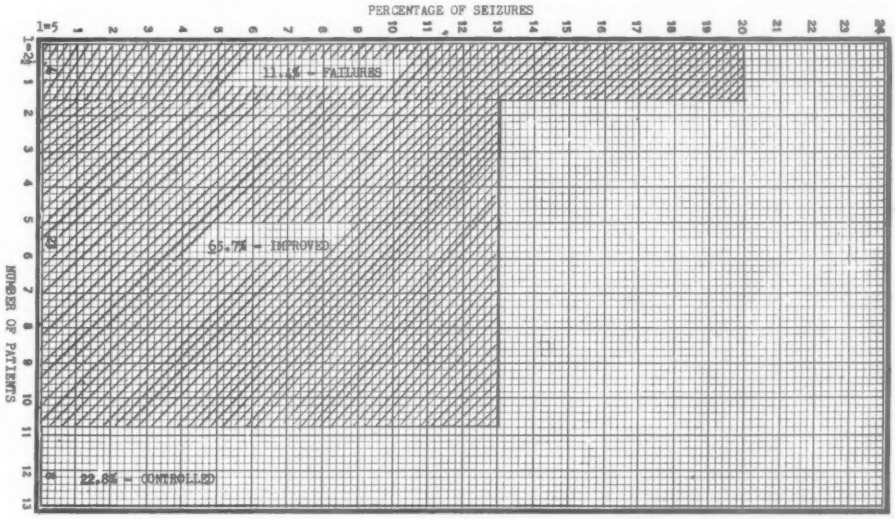
Do you feel that there is improvement in your condition since treatment here? _____

dination, dizziness, marked gum hyperplasia, reduction of weight, heightened irritability and restlessness. In many cases,[8, 12, 23, 24] the combining of phenobarbital with diphenylhydantoin sodium was found remarkably more efficient than the independent use of either drug in reducing seizures and, more gratifying, the deleterious effects such as mentioned above were frequently diminished.

Of no negligible importance is the proving of 3 methyl-5, 5-phenylethylhydantoin (whose trade name is "Mesantoin") combined with phenobarbital,* which are the findings of Loscalzo,[18] who indicated that the use of this drug reflected a 60% reduction in grand mal seizures among 67 cooperative patients, the majority of whom had epilepsy of undetermined causes. Among his conclusions drawn from a 3 year study we find that the toxicity of Mesantoin is inordinately low, only one patient having been unable to take the drug because of rash. Despite the encouraging results in reduction of grand mal seizures, however, it must be noted that this medicant did not improve patients suffering from petit mal and psychomotor disturbances.

It is well worth observing that Clein[6] and Kozol[16] also indicate in their findings regarding the use and efficacy of Mesantoin that a small amount of phenobarbital increases the anticonvulsant action of same. (A combination of 3-methyl-5, 5-phenylethylhydantoin and phenobarbital is now distributed under the name Hydantal). It is interesting to note that Kozol,[16] while recording also that 60% of the patients on this drug therapy were greatly improved, he differed from Loscalzo,[18] in that his opinion of psychomotor and focal attacks were also included in improvement of grand mal reduction. The papers published by other workers[5, 7, 9, 13, 25] afford us with a balanced number of studies from which to draw conclusions on the relative efficacy of Hydantal, diphenylhydantoin sodium, and Mesantoin alone in the treatment of epileptic seizures.

It is appropriate to note that Loscalzo,[18] in listing side effects, encountered gingival hypertrophy in 6% of patients on Hydantal therapy, 4½% suffering skin rash and 15% experiencing slight drowsiness. This rash was intensely pruritic and Kozol,[16] in his first studies, indicated a 10% incidence in his patients until he initiated small doses at the start, gradually increasing them, whereas, it was then found that virtually no rashes resultantly appeared. The only significant clinical finding in the cases of rash studied by Loscalzo[18] was a moderate leukocytosis.



However, warning must be given that dangerous complications may ensue in some patients taking this type of drug, as indicated by Bloom, Lynch and Brick,[4] regarding Mesantoin poisoning with aplastic anemia. The report on this case revealed that a white woman of 34 years, suffering grand mal seizures, developed severe aplastic anemia with hyperplastic bone marrow as a result of sensitivity to Mesantoin. The patient recovered, however, after

Case	Initials	Age	Sex	Frequency of Seizures	Onset Age	Duration Years	Type of Seizures
1	J. M. W.	25	M	2 or 3 times monthly	13	12	Grand Mal
2	A. F. L.	32	M	once every 8 months	19	11	" "
3	H. C. K.	30	M	3 times daily	17	13	" "
4	W. C.	37	M	once every 6 weeks	15	15	" "
5	M. H.	26	M	once every 2 weeks	20	6	" "
6	L. M.	26	M	once every 2 weeks	22	4	" "
7	H. D.	19	M	once every month	18	1	" "
8	H. D.	40	M	once every 2 weeks	22	18	" "
9	H. L.	27	M	once a year	18	9	" "
10	L. K.	18	M	once every 10 days	14	4	" "
11	G. B.	26	M	once monthly	25	1	" "
12	F. S.	27	M	once weekly	25	2	" "
13	G. D.	21	M	3 times yearly	17	4	" "
14	J. J.	25	M	1 or 2 times monthly	19	6	" "
15	J. H.	19	M	once every 2 months	19	11 mo.	" "
16	T. S.	48	M	3 or 4 times monthly	20	28	" "
17	J. E. B.	26	M	once weekly	23	3	" "
18	R. W.	41	M	two monthly	6	35	" "
19	I. H.	19	M	two monthly	19	10 mo.	" "
20	E. J. B.	26	M	once monthly	12	14	" "
21	L. P.	23	M	2 or 3 times weekly	19	4	" "
22	W. C. S.	43	M	1 each day or night	39	4	Hysteria
23	J. W.	26	M	2 or 3 times monthly	8	18	Grand Mal
24	M. L. A.	35	M	3 or 4 times yearly	10	25	" "
25	S. J.	42	M	1 or 2 times weekly	42	11 mo.	" "
26	W. P.	27	M	irregular intervals	9	18	" "
27	C. G.	34	F. M.	irregular intervals	11	23	" "
28	A. C.	24	M	irregular intervals	18	6	" "
29	E. C.	23	M	two weekly	13	10	" "
30	H. H.	23	M	3 times weekly	21	2	" "
31	W. Y.	24	M	one weekly	14	10	" "
32	W. C.	37	M	once every 6 weeks	23	14	" "
33	A. B.	24	F. M.	once monthly	13	11	" "
34	E. L.	21	F. M.	weekly intervals	13	8	" "
35	M. P.	19	M	two weekly	13	6	" "

Diagnosis	Previous Medication	Results	Present Medication	Results	Remarks
Psychomotor	Unknown		Hydantal	Improved	
Idiopathic	Unknown		Hydantal	Improved	
Focal	Unknown		Mesantoin	Improved	
Focal	Phenobarbital	Moderate Improvement	Mesantoin	Controlled	
Focal	Dilantin	Moderate Improvement	Mesantoin	Controlled	
Idiopathic	Phenobarbital	No marked Improvement	Mesantoin	Controlled	
Focal	Phenobarbital	No marked Improvement	Mesantoin	Markedly Improved	
Focal	Phenobarbital	No marked Improvement	Mesantoin	Controlled	There is epilepsy in the family
Focal	Dilantin	Some Improvement	Mesantoin	Controlled	There is epilepsy in the family
Idiopathic	Unknown	Some Improvement	Mesantoin	Controlled	Mother has epilepsy
Idiopathic	None		Mesantoin	Controlled	
Focal	Unknown	Some Improvement	Mesantoin	Improved	
Focal	Phenobarbital	No marked Improvement	Hydantal	Improved	Mother and G. M. had epilepsy
Focal	Phenobarbital	No marked Improvement	Mesantoin	Improved	
Idiopathic	Unknown		Mesantoin	Improved	
Focal	Dilantin	No marked Improvement	Mesantoin	Markedly Improved	Brother has epilepsy
Idiopathic	None		Hydantal	Markedly Improved	
Idiopathic	Dilantin and Phenobarbital	Satisfactory	Hydantal	Improved	
Idiopathic	Unknown		Hydantal	Controlled	
Focal	Dilantin and Phenobarbital	Satisfactory	Hydantal	Improved	Mother died of epilepsy
Focal	Phenobarbital	Slight Improvement	Mesantoin	Improved	
Hysterical Epilepsy	Unknown		Mesantoin & Hydantal	Failure	
Focal	Dilantin and Phenobarbital	Some Improvement	Mesantoin	Improved	
Focal	Many Rx Incl. Mesantoin	Poor	Hydantal	Failure	Mother and Sister have epilepsy
Idiopathic	Mesantoin Dil. & Phepo.	Improved	Mesantoin	Improved	
Idiopathic	Phenobarbital	Improved	Mesantoin	Improved	Father had epilepsy
Idiopathic	Various Rx	Little Improvement	Mesantoin	Failure	Aplastic anemia
Psychomotor	Unknown		Mesantoin	Improved	
Idiopathic	Unknown		Mesantoin	Markedly Improved	Mother was epileptic
Focal	Dilantin Phenobarbital	Some Improvement	Mesantoin	Improved	
Focal	Phenobarbital	Some Improvement	Mesantoin & Hydantal	Failure	The sequelae became milder
Focal	None		Hydantal	Markedly Improved	
Idiopathic	Dilantin	Failure	Mesantoin & Hydantal	Improved	
Idiopathic	Phenobarbital	Some Improvement	Mesantoin	Improved	Cousin is epileptic; patient is mentally deficient
Psychomotor	Dilantin	Some Improvement	Mesantoin	Improved	

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numerous transfusions and the institution of a regimen of penicillin, streptomycin and various hematinics. Monthly hematologic studies should, therefore, be made when indicated in order to recognize and take prompt steps to control and circumvent the above mentioned condition.

It is interesting to study and compare the respective conclusions of investigators in the field of epilepsy therapy in various forms with Dilantin, phenobarbital, Mesantoin and Hydantal. Von Hagen,[25] using Hydantal in 16 cases of epilepsy, indicated that in agreement with other workers, it was most effective in controlling grand mal seizures, the toxic effects being in his opinion negligible. It is surprising to note that certain distinct differences in results are gained by observers using Mesantoin and phenobarbital. Otto,[13] agreeing with studies by Kozol,[16] states that Mesantoin is of "great benefit in the treatment of psychomotor epilepsy," while Loscalzo,[18] reports adversely that petit mal and psychomotor epilepsy were not improved with Mesantoin and phenobarbital. It may be of importance, however, to note that Otto[13] specialized in the treatment of psychomotor seizures, whereas 45 of the 67 patients of Loscalzo[18] suffered from grand mal, the remaining 22 patients being in the categories of psychomotor (12), petit mal (5), focal convulsions (3), myoclonic (1) and Jacksonian (1).

Fetterman and Friedman, in collaboration with Weil and Victoroff,[9] in their 1947 report regarding the treatment of 50 patients for one year with Mesantoin, among whom the majority were diagnosed as idiopathic epileptics, arrived at the following conclusions: That Mesantoin, like Dilantin, is valuable for grand mal and Jacksonian attacks, and not for petit mal; that, though effective alone, its major role is seen as being a supplement to Dilantin sodium; that "Mesantoin has proved more effective than phenobarbital alone and compares quite favorably with Dilantin sodium. Even if its anti-convulsant properties may be less than those of Dilantin, its side actions are milder and larger doses can be safely tolerated."

The findings of the writer in a study of 35 consecutive cases treated, during a follow-up of 1 to 2 years in private practice and at the Virginia State Penitentiary, bear out the conclusions arrived at by the above mentioned workers in the field of epilepsy that Mesantoin is a drug progressively acquiring a reputation for dependability and relative safety from toxicity, in controlling grand mal epilepsy.

At the outset it must be indicated that the above study was undertaken after a complication in therapy with Mesantoin had taken place,[4] in spite of good results accomplished by other workers. As is expected, prison facilities are usually limited, rendering it difficult to carry out a thorough scientific project in epilepsy. We do not have an electroencephalograph or a laboratory personnel with high standards of training as found in various hospitals, since all our technicians are inmates. This does not, however, detract from the standard conception that a drug's value is considerably enhanced if it can be administered without a great deal of laboratory procedures. Unless indicated, routine blood counts were not taken, electroencephalographic recordings were eliminated, and the symptomatology presented is entirely subjective in nature. Though one is aware of the limitations of this paper, the lack of complications noted during a follow-up of 1 to 2 years among the prison patients served to stimulate the reporting of the cases mentioned below.

Before deciding on anti-convulsive therapy the questionnaire entitled "seizures" was completed. Mesantoin in an average daily dose of 3 grains or Hydantal in similar doses were given. This treatment was limited to grand mal seizures evidenced in idiopathic, focal, psychomotor and hysterical epilepsy. These diagnoses were made mostly on the basis of the history presented and some error in same is therefore anticipated.

SUMMARY, CONCLUSION

I. 35 Patients were treated with Mesantoin or Hydantal and studied during a period of from 1 to 2 years. These averaged 98 seizures per month prior to the above therapy.

II. Of this group 17 presented symptoms of focal epilepsy, 14 of idiopathic, 3 of psychomotor and 1 of hysterical epilepsy.

III. The symptoms of 8 patients (22.8%), completely controlled by this drug, occurred equally in the focal and idiopathic types.

IV. After treatment with Mesantoin and Hydantal 22 patients showed a reduction in seizures by 31.6%.

V. The patients whose symptoms were not improved by this drug stated that sequelae of seizures were much milder.

VI. One patient developed aplastic anemia but recovered.

VII. No toxic reactions were noted among the 31 prisoner patients treated.

Mesantoin and Hydantal, though not necessarily superior, are drugs which have demonstrated a safe efficacy in many respects in the treatment of epileptic patients. Their toxicity among the Penitentiary patients was not observed.

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*Mesantoin and **Hydantal were generously supplied by the Sandoz Chemical Works, Inc., N. Y., for the purpose of this study.

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VASOPRESSOR DRUGS IN SPINAL ANESTHESIA

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A study of some of the vasopressor drugs currently available for spinal anesthesia was undertaken in order to make a comparison of their effectiveness. Significant decrease in blood pressure may be expected during spinal anesthesia when no vasopressor drug is used, and consequently employment of such drugs obviates the difficulties of hypotension which deter use of spinal anesthesia.[2]

A number of drugs may be used to sustain blood pressure during anesthesia, but ephedrine is probably the one most commonly used. Ephedrine was employed in our study, as well as methedrine† (desoxy-ephedrine) and in a few cases oenethyl‡ (menthyl-amino-heptane).

Method—Spinal anesthesia was administered to patients selected as to applicability of this type of anesthesia to the patient and the operative procedure. In 124 instances the continuous spinal technique was used, in 148 instances a single injection of the anesthetic drug was administered, and all anesthetics were given by medical anesthetists.

The drugs used for spinal anesthesia were procaine, pontocaine, a mixture of procaine and pontocaine or pontocaine and 10 per cent dextrose. The dosage of the drugs was varied to suit the type of operative procedure but was kept within accepted safe limits.

In all except the most minor cases, administration of intravenous fluid was started immediately after the anesthetic in order to sustain blood pressure and to allow for rapid administration of subsequent doses of the vasopressor drug if necessary, as well as transfusion when indicated. All the patients in our study were given sedatives and either atropine or scopolamine preoperatively. During operation adequate sedation was maintained by intravenous administration of nembutal or morphine and scopolamine or small amounts of sodium pentothal.

Each vasopressor drug was given to consecutive cases or to every other case in most instances, and there was no selection of special patients on whom to use any one drug. The drugs were administered at the time of spinal puncture, and therefore almost simultaneously with onset of anesthesia, by the single injection technique, and about ten minutes before onset of anesthesia by the continuous spinal technique. Ephedrine was given in a dose of 25 to 50 mg., methedrine in a 10 to 20 mg. dose, and oenethyl in a 50 or 75 mg. dose.

Blood pressure was determined by auscultation over the brachial artery before administration of the vasopressor drug at onset of anesthesia, and at five-minute intervals during the operative procedure the estimations of blood pressure, pulse rate and respiration were recorded.

TABLE 1

Operation	*Number of Cases*
Amputation of leg	17
Amputation of penis	1
Amputation of prolapsed rectum	1
Aneurysmorrhaphy	2
Appendectomy	22
Cesarean section	57
Cystotomy, suprapubic	4
Debridement of ulcer	3
Dilatation and curettage	1
Dilatation and curettage and postperineorrhaphy	1
Epididymectomy	1
Evacuation of hematoma	2
Excision of anal fistula	1
Exploration of bile ducts	1
Fusion, spinal	2
Gastrectomy	1
Graft, bone	2
Graft, skin	1
Hemorrhoidectomy	14
Hernioplasty	19
Hysterectomy	19
Hysterectomy, vaginal, and anteroposterior repair	2
Hysterotomy and bilateral tubal ligation	2
Incision and drainage	1
Laparotomy, exploratory	11
Ligation of common iliac artery	1
Ligation of vein	2

*Removed to St. Thomas Hospital, Nashville, Tenn.
**From Department of Anesthesia, Medical College of Virginia, Hospital Division, Richmond, Va.
†Supplied through courtesy of Burroughs Wellcome & Co., Tuckahoe, N. Y.
‡Supplied through courtesy of Bilhuber-Knoll Corp., Orange, N. J.

Nephrectomy	3
Oöphorectomy and salpingectomy	2
Osteotomy	1
Prostatectomy	9
Removal of cervical stump, La Force operation, and perineorrhaphy	1
Removal of ruptured intervertebral disc	3
Repair of jejunal loop	1
Resection, abdominoperineal	5
Resection of colon	10
Resection of diverticulum of duodenum	1
Resection of pancreas	1
Resection, transurethral	5
Salpingectomy, bilateral, partial	7
Salpingoöphorectomy, anteroposterior repair and appendectomy	20
Spermatocelectomy	2
Sympathectomy	9
Thrombectomy and bilateral sympathectomy	1
TOTAL	272

Results—In determining the value of the various vasopressor drugs that were studied, it was decided to regard as significant a decrease in blood pressure of 25 per cent from the initial level. Table 2 lists the number of such cases with each vasopressor and also the percentage of cases receiving an additional dose of the drug.

With methedrine a fall in blood pressure of 25 per cent occurred in 14 cases (12.2 per cent), in 10 (8.7 per cent) of which it was necessary to administer additional vasopressor. When ephedrine was used, 27 cases (25.4 per cent) exhibited a comparable drop in blood pressure, and 19 (17.9 per cent) received an additional dose of the drug. In the small number of cases in which oenethyl was used, the designated drop in blood pressure occurred in 4 (18.1 per cent), with the same number, 4 (18.1 per cent), requiring an additional dose.

DISCUSSION

Factors influencing decrease of blood pressure under spinal anesthesia are initial level of blood pressure, level of spinal anesthesia, type of surgery, length of operation, and age and general condition of the patient. The anesthetic drug may also be a factor, but in this study no estimation was made of the effect of the drugs in question. However, the various operative procedures as well as variations in physical status of the patients were quite uniform in each of the groups. Spinal anesthesia was not administered to patients whose condition did not warrant its use.

The fact that there were no alarming falls in blood pressure in these cases indicates that all the vasopressor drugs that were used had considerable effectiveness. Of the three drugs studied in our series of cases, methedrine was the most effective, which corroborates previous reports.[2,7]

In 30 of our cases use of a vasopressor drug was not considered necessary because the level of anesthesia was kept low, although later its administration was deemed advisable in 20 per cent of this number.

The pharmacology of the drugs differs somewhat, indicating that one drug might be more applicable in one patient than in another.

Use of ephedrine produces a rise in blood pressure largely by increasing cardiac output because of stimulation of the myocardium.[5] It also causes arteriolar constriction, and the stimulatory effects of ephedrine on the central nervous system are well known. Therefore, employment of this drug for patients in whom myocardial strain is already manifest might be open to question and should certainly be done with circumspection.

The rise in blood pressure following administration of oenethyl is largely due to arteriolar constriction, although some stimulation of the myocardium does occur because of stimulation of the sympathetic nervous system.[4] Experimental use of oenethyl has demonstrated that it is much less likely to cause dis-

TABLE 2

Vasopressor Drug	Number of Cases	Cases with 25% Fall in Blood Pressure		Cases Given Additional Vasopressor Drug	
		Number	Per Cent	Number	Per Cent
Methedrine	114	14	12.2	10	8.7
Ephedrine	106	27	25.4	19	17.9
Oenethyl	22	4	18.1	4	18.1
None	30	6	20.0	6	20.0

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turbance of cardiac automaticity than is either adrenalin or ephedrine,[3] which fact may be of importance when it is necessary to supplement the spinal anesthetic with a general anesthetic, especially cyclopropane.

A similar lack of disturbance of cardiac rhythm by methedrine has been reported.[6] Methedrine obtains its circulatory effect chiefly by peripheral vasoconstriction, but large doses act directly on the myocardium.[2] Originally methedrine was used to stimulate the central nervous system, and Dodd and Prescott, who in 1943 were the first to suggest its use as a peripheral vasoconstrictor, observed that its duration of action was longer than that of other vasopressor drugs.[1]

CONCLUSIONS

1. Use of vasopressor drugs is of considerable value in sustaining blood pressure during spinal anesthesia.

2. Of the three drugs studied in our series of 272 cases, methedrine was the most effective.

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Hepatitis is Occupational Hazard.

A liver infection called hepatitis has proved to be a definite hazard to medical personnel working with blood and blood derivatives, two Memphis physicians write in the March 31 *Journal of the American Medical Association*. Drs. Merlin L. Trumbull and D. J. Greiner, said the disease can be transmitted by minute quantities of infected blood or serum introduced into the system through a small opening or scratch in the skin.

The doctors believe hepatitis among medical personnel may increase. They attribute this possible increase to the expanding use of blood and its derivatives. They reported that in a survey of occupational diseases among the medical personnel in two of the hospitals mentioned in the report, hepatitis was the most common during this three-year period.

According to the article, the best protection against this occupational form of hepatitis at present is strict adherence to the rules governing the handling of any diseased or infected substance. Wearing gloves, careful sterilization of all instruments, especially those penetrating the skin, marking bottles containing known infected blood, and avoidance of spilling blood on the hands were a few precautions mentioned

DR. JOHN J. CRAVEN, PHYSICIAN TO JEFFERSON DAVIS*

Chester D. Bradley, M.D.,
Newport News, Virginia.

The two most important political captives of the nineteenth century were Napoleon Bonaparte and Jefferson Davis. Each had a physician assigned to him by his captors. Napoleon's medical attendant was Dr. Barry E. O'Meara, an Irish surgeon in the British Navy. Jefferson Davis' medical attendant was Dr. John J. Craven, Chief Medical Officer at Fort Monroe, Virginia. Both O'Meara and Craven wrote books about their famous patients and thus achieved a reflected celebrity. O'Meara's account of his association with Napoleon on the island of St. Helena lifted him from obscurity. Jefferson Davis' physician, however, had already achieved individual distinction, not as a medical man, but as an inventor, for the healing profession was only Craven's second love.

John Joseph Craven was born in Newark, New Jersey, on September 8, 1822. His parents were very poor and early in life he was apprenticed to a carpenter. Craven had very little schooling, his education being largely obtained through reading and observation. After helping to build the Passaic Chemical Works, he accepted a position in the plant as superintendent of construction and repairs. The products of this plant were acids and dry salts and from a study of the processes used in their manufacture, the young man acquired a taste for science.

In 1845 when Morse's telegraph line from Philadelphia to New York was being built across New Jersey, Craven left the chemical plant and went to work in the construction department of the Magnetic Telegraph Company. The most important problem in telegraphy in those days was to get the wires over large bodies of water, all attempts at insulation having been without success. In 1847 Craven was shown a newly introduced substance called gutta percha, the gum of a Malayan tree. Craven covered a copper wire with gutta percha and tested it under the surface of Bound Creek near Elizabeth, New Jersey, that same year of 1847. Satisfied with the result, he obtained permission from his employers to submerge his wire in the Passaic River at the railroad drawbridge at Newark, connecting it with the land wires on each side. The submerged gutta-percha-insulated wire conducted electrical impulses without escape (Reid).

On June 15, 1848, Craven laid a cable for the Magnetic Telegraph Company uniting New York and Jersey City, and making complete the line between New York and Washington. This cable extended from the Cunard dock in Jersey City to a dock at the foot of Cortlandt Street. In a few days, however, it was destroyed by an anchor, and a less exposed situation was selected. Craven applied for a patent on his invention, but it was refused on technical grounds. Bitterly disappointed, the young inventor left the telegraph company and sailed around the Horn to California with a party of young men from Newark in a boat they had purchased.

Apparently, Craven did not have much success as a gold-hunter, for he was back in Newark in 1851. He now settled down to the more serious business of studying medicine, placing himself under the preceptorship of Dr. Gabriel Grant of Newark. He also attended lectures at the College of Physicians and Surgeons in New York City. When the Civil War broke out, Craven, who was practicing medicine in Newark, joined the Union Army as a surgeon. He took part in various expeditions against Confederate-held points on the South Atlantic Coast, being cited for his conduct in the assault on Fort Pulaski, Georgia. Craven rose steadily through the grades to be Medical Director of the 10th Corps. Early in 1865 he was made Medical Purveyor of the Department of Virginia and North Carolina with headquarters at Fort Monroe, Virginia, where he also filled the post of Chief Medical Officer with rank of brevet lieutenant-colonel.

Jefferson Davis was brought to Fort Monroe for confinement in May, 1865. The Confederate President was execrated and feared by his captors even more than Napoleon had been in 1815. Many of Davis' foes hoped that Fort Monroe would be his "living tomb," as St. Helena had been that of Bonaparte. Fort Monroe, the mightiest fortification in the Western Hemisphere in 1865 was considered the only

*Read before the medical staff of Riverside Hospital, Newport News, Virginia, October 13, 1950.

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secure place to keep the indomitable Jefferson Davis. It is situated at the very tip of Old Point Comfort, a narrow strip of sandy soil projecting about two miles out into Chesapeake Bay. Its thick granite walls rise 35 feet above a water-filled moat which ranges from 75 to 100 feet in width and surrounds the fort in its entirety. The fort has three gates, or sally-ports, which are approached by bridges over the moat like a medieval castle. Designed by one of Napoleon's marshals in exile (Simon Bernard), Fort Monroe was built to accommodate 412 cannon. Not all of these guns had been mounted, however, and some of the gun rooms (casemates) in the walls of the fort had long been used as living quarters. It was in a casemate of the second front of the fort that Jefferson Davis was held in solitary confinement for four and one-half months (May 22 to October 2, 1865). Through Dr. Craven's intercession, the prisoner was then moved to Carroll Hall, a two-story brick building which formerly stood in the northwest bastion of the fort, where he spent the balance of his two-year imprisonment.

The story of Dr. Craven's relationship with Jefferson Davis is as well known as that of O'Meara with Napoleon. The kindly and diplomatic Craven gradually succeeded in persuading General Nelson A. Miles to remove the more severe restrictions on the imprisoned Confederate chief. However, Craven, like O'Meara, eventually grew so sympathetic with his prisoner-patient that he came into open conflict with the authorities and had to be removed in December 1865 after only seven months of attendance on Jefferson Davis. Dr. O'Meara had managed to stay with Napoleon about three years, but he undoubtedly would have been removed earlier if the isolation of St. Helena had not made his replacement difficult. Napoleon himself was never to leave his island-prison alive, for he died of cancer of the stomach in 1821. Jefferson Davis, as is well known, fared much better, for he was released from Fort Monroe in 1867 on a bail bond signed by Horace Greeley. He lived to the ripe age of 81 years, dying at New Orleans, Louisiana, in 1889 of bronchopneumonia.

Craven, mustered out of the service on January 27, 1866, returned to Newark where he gave further evidence of his versatility by serving as postmaster of that town. He also published a book that same year of 1866 entitled, *The Prison Life of Jefferson Davis.* This book was based on a diary and notes which he had kept of his experiences and conversations with Jefferson Davis in emulation of Dr. O'Meara*. In fact, the title page of Craven's book bears a quotation from O'Meara's book about Napoleon, reading as follows: "Had I died on the throne, enveloped in the dense atmosphere of power, I should to many have remained a problem. Now, misfortune will enable all to judge me without disguise."

DR. JOHN J. CRAVEN
A photograph he sent to Mrs. Jefferson Davis.
(Courtesy, Confederate Museum, Richmond)

*Barry Edward O'Meara (1786-1836) came of a prominent Irish family. He studied anatomy in Dublin and surgery in London. Dismissed from the army in 1807 for participating in a duel, he became a surgeon in the British Navy. In 1815 he was assigned to act as Napoleon's physician on the island of St. Helena, remaining till 1818 when he was sent back to England for being too sympathetic with the prisoner. In 1822 he published a book "Napoleon in Exile; or A Voice from St. Helena," which was based on his diary. O'Meara was eulogized by Lord Byron in the third stanza of "The Age of Bronze": "And the stiff surgeon, who maintained his cause, Hath lost his place and gained the world's applause." He died in London of "erysipelas in the head." (*National Dictionary of Biography.*)

Dr. Craven visiting Jefferson Davis during his imprisonment in the casemates of Fort Monroe.
(From 1905 edition of Craven's book)

Craven's book, like that of O'Meara, had a large sale. It was praised by some and condemned by others, as would be expected from the controversial nature of the subject. The New Jersey physician favored a policy of conciliation toward the conquered Southern States and he thought that his book might serve as a guide. Craven writes, "Believing that the views of Mr. Davis may throw important light on the true policy to be pursued, the author noted down all such as he could remember, or had made notes of, as faithfully and as conscientiously as if giving his evidence under oath in a court of justice."

The authenticity of Craven's book has been impugned (Current Literature, June 1905). It has even been suggested that the book was invented by a professional writer. However, let it be noted that the events in Craven's book can be corroborated step by step, with startling accuracy, in the *Official Records of the Union and Confederate Armies, Series II, Volume VIII*. This official compilation of all documents relating to prisoners of war was not published until 1899, thirty-three years after Craven's book. (In 1905 Craven's son, William Darcy Craven, published a reprint of *The Prison Life of Jefferson Davis.*)

Critics on both sides of the Mason-Dixon Line have challenged the accuracy of some of the details of Craven's description of the shackling of Jefferson Davis. However, Craven states most clearly in his book that ***he himself did not witness this event*** and that his account of it "has been based on the evidence of others who came fresh from the scenes pictured." The shackling occurred on May 23, 1865, whereas Craven was not assigned to attend Jefferson Davis until the next day, May 24. At this point Craven says, "I now reach the commencement of my personal relations with the prisoner, and for all that follows am willing to be held responsible."

On May 25 Craven courageously went to General Miles and asked him to remove the shackles from Jefferson Davis. This was not done until May 28, and when it was done, Craven naturally took credit for it. However, it is now apparent that General Miles removed the shackles only because he was ordered to do so by a telegram from the Secretary of War on May 28. Not being in the confidence of Gen-

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eral Miles, Craven knew nothing of this telegram. Craven, on his visits to the Davis casemate, had always to be accompanied by the Officer of the Day, and it is significant that in nearly every instance the doctor gives the name and rank of the officer accompanying him and usually states the approximate hour or portion of the day in which the visit occurred. That Craven's book was based on his diary seems indisputable. That Dr. Craven, not a writer himself, may have collaborated with a professional writer in the production of this book, does not at all invalidate it.

In 1867 Craven moved to Jersey City where he resumed the practice of medicine. Here he was approached by the officials of the Communipaw slaughter house which had been declared a public nuisance. They appealed to Craven for help with their problems because of his knowledge of chemistry and sanitation. The inventor in Craven was reawakened and he devised processes and invented machinery which enabled the abattoir to dispose of its waste matter by converting it into salable by-products. He thus acquired the standing of an expert in the problems of slaughter houses, being consulted by firms throughout the country. Attacking the problem of the preservation of fresh meat, Craven invented a refrigerating apparatus for use in cold storage, and for transportation of meat in railroad cars and steamships. The first vessel to be fitted out under Craven's supervision was the Cunard steamer "Abyssinia" in 1876.

Craven, the middle-aged physician-inventor, was more fortunate with his patents than he had been with his youthful invention of gutta-percha insulation. Wealth came to the doctor and in 1881 he retired from medical practice, moving to Patchogue, Long Island, where he henceforth devoted his attention to his business interests. With characteristic energy, Craven took a large share in the life of his adopted community: President of the Board of Education, President of the Board of Health, President of the Library Association, Fire Commissioner, and even President of the Cemetery Association! John J. Craven is remembered as having a decided taste for literature, especially versification, and remarkable memory enabling him to quote extensively from his favorite authors. He was fond of the theater, both Shakespearean and modern, and he also had an outdoor side for he enjoyed sports, fishing and hunting. Craven died of a cerebral hemorrhage on February 14, 1893, at the age of 70 years. He was buried at Patchogue.

Dr. Craven, gifted though he was, left no imprint on medicine. This is probably because Craven's aptitudes were mechanical and scientific, and the medical field at that time offered scant play for the talents of such a man. Medical practice, in Craven's day, was a rather vague art based on tenuous theories and relying on such crude measures as blistering, cupping, leeching, poulticing and purging. The great modern era of medical discoveries and advances did not dawn until Craven had retired from medical practice. Had Craven been born in this present century it is very likely that he would be an outstanding medical research scientist. One of his colleagues in the Union Army had this to say about him: "He is a very excellent, kind, skilful and conscientious medical attendant. A vigorous follower of field sports; rather spare of habit; pure Roman features, set off to advantage by military whiskers and moustache; a voice of excellent modulation, and manners of great courtesy and politeness. He is a devotee of science in all its branches, and more fully fills my idea of a savant than any other medical man it has been my fortune to meet in the army."

To Craven, the inventor, belongs the credit for devising the first successful submarine cable. To quote a speech made at the cable celebration in Philadelphia in 1858, "John J. Craven of Newark, New Jersey, made and laid the first practical, substantial, available submarine telegraph; and let his name stand out in its proper place."

After Craven's departure from the Magnetic Telegraph Company, the work he had begun was continued by others. The transatlantic cables of 1858, 1865 and 1866 were all insulated with gutta percha (Charles Bright). "The type of core . . . accepted by the Scientific Committee for the new cable was to consist of a strand of seven copper wires . . . *coated with four layers of gutta percha* . . ." (my italics).

Though John J. Craven derived no wealth from this invention of his youthful days, he at least had the satisfaction of seeing realized in his lifetime the enthusiastic prediction he made in 1848 when he wrote, "I have surely struck it. I have now in my possession a wire that is, in a short circuit beneath a stream, in connection with a main line, doing its work faithfully. Mark my words: It will yet cross rivers, bays, seas and oceans, and connect continents."

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U. S. Patent No. 131,429, September 17, 1872, for Rendering and Drying Apparatus in the Treatment of Animal-Matter, issued to John J. Craven.

U. S. Patent No. 175,939, April 11, 1876, for Refrigerating Chamber, issued to John J. Craven.

2914 West Avenue.

The Oath of Hippocrates

By Apollo, Aesculapius and Hygeia I declare,
By Panacea and all male and female gods, to whom I swear
That here I make my promise full and freely in their sight,
An oath to be upheld with all my knowledge and my might.
Him will I deem my father, who teacheth unto me
The mysteries of medicine; and with him, if need there be,
Will divide my last possession and for his children care
As were they my own brothers; and shall no effort spare
To instruct them in the glories of the gentle healing art,
And will take from them no recompense in doing thus my part.
To those who make a vow likewise of succour unto me
On me the same devolves in turn, a sacred claim to be.
I shall prescribe for sufferers the things that best avert
All harmful influences which may do them any hurt.
Prayers for abortions or for poisonings shall not reach my ear,
A stainless life my single aim, Humanity my sphere.
For calculus I will not cut, but leave that field alone
To those who mark that malady a province of their own.
Iniquity eschewing, I shall soothe the ailing throng,
Unshamed with slaves or freemen, doing no woman wrong.
No tales my tongue shall utter, no secrets shall betray,
But lofty faith and honour shall I strive for, day by day.
And if this vow and promise I maintain intact and sure,
My present shall content me, my future stand secure;
But should I soil my honour and break this sacred vow,
May I to perjury's penalty my head ignobly bow.

Theodore C. Merrill, M.D.
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A STUDY ON THE CORRELATION BETWEEN DEMENTIA PRAECOX AND THE MONTH OF BIRTH

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It has been stated by J. de Sauvage Nolting in 1934[5] and again in 1939[6] that in an analysis of his cases of dementia praecox for the month of birth, he found apparently a larger number of these patients were born in the winter months than at any other time. This unusual fact was related by L. Bellak[1] in his work, "Dementia Praecox". Bellak continued in some detail and noted ". . . that the lowest incidence apparently was in June, the difference of incidence between March and June being quiet significant. Only 153 of the large group were born in June; 317, the highest incidence, was in March." He further stated that . . . "any confirmatory or negating data should be easy to find in any large state hospital".

With the cooperation of Miss Edna Lantz, statistician for the Department of Mental Hygiene and Hospitals of Virginia, it was possible to tabulate as of June 30, 1949, the month of birth of patients resident in all four of the Virginia state hospitals classified by dementia praecox diagnosis.

There were 1469 patients with the birth date coded —that is, those patients whose complete birth dates were recorded. There were 1881 patients with the birth date uncoded. The former group was used for this survey. Of this group of 1469 patients, 739 were born between the winter months of October 1 to April 1. This is 50.3%. In other words, only 0.3% more were born in the winter than in the summer months; this is an insignificant and negligible amount.

It was felt that the various sub-types of the dementia praecox classification should also be included in this survey to see whether any one or more types might vary from this average. Under the simple type it was found that 79 out of 149 or 53% were born in the winter months. Under the hebephrenia type, 197 out of 389 or 50.9% were born in the winter months. Under the catatonic type, 172 out of 355 or 48.4% were born in the winter months. Under the paranoid type, 264 out of 531 or 49.7% were born in the winter months. Under the classification "other types" it was found that 23 out of 45 or 51.1% were born in the winter months. Thus it seems that none of the subtypes vary sufficiently from the over-all average to be called outstanding.

As to the findings of the birth dates in March and June—this writer found 117 patients of the group of 1469 patients being born in March and 125 patients of the group of 1469 patients being born in June.

Since the winter months comprised October 1 to April 1 and the summer months comprised April 1 to October 1, it was felt that this might represent too much "spread". So the winter months were restricted from December 1 to March 1 and the summer months from June 1 to September 1. These results, representing 25% of the year, represented 399 patients out of 1469 or 27.1% for the winter. For the summer, again of course representing 25% of the year, the results revealed 340 patients out of 1469 or 23.1%. The 2% difference seems minimal as the percentages approached 25% so closely.

However, to be statistically accurate, *the complete birth rate by month should be determined in the same year as was that of the patients in the survey. The age on first admission varied from 15 to 50 approximately, so the natality percentage per month could be any year from 1890 to 1925. It is observed in reviewing the statistics **that the crude birth rate per 1000 population per month in any one year varies up to 12% at the most and with no definite monthly regularity—with the exception of October, November and December, which often have the lower rates. This is for the total population, however, and not just Virginia.

The figures on birth rates for Virginia are not available for the actual period involved with the patients in this survey. We do have the statistics for the year 1947, however, which will be used as a

*I am indebted to Dr. Stanley Williams, Professor of Psychology, College of William and Mary, Williamsburg, Virginia, for statistical guidance.

**Vital Statistics Rates in The United States 1900 to 1940, Page 668. United States Department of Commerce, Bureau of the Census.

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sample.*** Of a total of 82,141 people born in Virginia in 1947, about 1/12 or 8.33% should be born monthly. In the three winter months of December, January and February, the rates were respectively 7.59%, 9.02% and 8.27% or a total of 24.88%. In the six winter months of October, November, December, January, February and March, the rates were respectively 8.26%, 7.73%, 7.59%, 9.02%, 8.27% and 9.15% or a total of 50.02%. In the three summer months of June, July and August the rates were respectively 8.15%, 8.46% and 8.65% or a total of 25.26%.

While seasonable variations vary at the extreme of 12%, the figures included in this study are well within this range and any small difference may well be within the range of sampling error.

On the surface it seems unusual (to say the least) that a person born in a winter month is more likely to have dementia praecox than one born in a summer month. Yet, such is the finding of J. de Sauvage Nolting. The findings in this paper do not substantiate the views of the above author. Yet, there are certain possibilities that must be taken into consideration to favor the above author's views. First, from the psychological viewpoint, it must be considered that the winter baby is much more of a hardship for the parents. It requires more care than the summer baby in food requirements, heat requirements and often medicinal requirements. There is then frequently tension—at the least—and even surliness on the part of mother, father and siblings with resultant displaced hostility toward the baby. Then, too, to be considered is the fact that since the winter is such an inconvenient season to have the baby, those babies that are born then are not planned babies. Frequently babies that are not planned are not wanted. This psychological factor should not be underestimated. Second, from the organic viewpoint, there are several factors different in the winter than in the summer. One such factor as suggested by G. L. Jones*, Superintendent, Eastern State Hospital, is the possible deficiency of a specific vitamin —namely, Vitamin D. As one considers the seasonal variations in food, other items of a possible deficiency easily come to mind. Another organic factor is the "stress and strain" evolved from the body's temperature regulating mechanism being forced to modify itself to vigorous meteorological environmental conditions. R. G. Hoskins[2] noticed a sluggish response of the temperature regulating mechanism in dementia praecox. Also to be included in this organic viewpoint is the footnote to a paper by Emil T. Hoverson[3]. He says ". . . The data secured from thousands of cases tend to show that the time of conception is quite an important thing in determining the chemical stability orginally." He was discussing the meteorologic climatic conditions as influencing chemical stability and believes this has a marked influence in the origins of dementia praecox.

The above two psychological factors and the organic possible deficiency can be eliminated definitely from any consideration as determining the origins of dementia praecox as occurring in the winter, for I believe this writer's statistics have eliminated the seasons as having any bearing on those possible factors on births in dementia praecox. However, the possible factors involved in meteorological conditions such as temperature, humidity, etc., have not been reasoned with adequately. The patients used for the statistics of J. de Sauvage Nolting in The Netherlands might have experienced much different climatic conditions at birth (or at conception) than those patients admitted to the Virginia state hospitals.

Since we are discussing winter versus summer, the climatic conditions existing in The Netherlands should be compared to the conditions in Virginia. First, the latitude is roughly between 52° and 54°N. in The Netherlands and between 36.5° and 39.5°N. in Virginia. Eastern and central Virginia are just a few feet above sea level. Western Virginia is variable—with the Appalachian Mountains occupying a large sector at an elevation of 3000 to 6000 feet. Some of The Netherlands is below sea level with the remainder but a few feet above sea level. Eastern and Central Virginia are quite humid. Western Virginia is less humid. The humidity in The Netherlands is not extreme. The heat in central and eastern Virginia is often uncomfortable in the summer. The winters are more tolerable. The stress and strain of "just living" thus occurs in the summer. This is not true for western Virginia where the summers are delightful, but the winters are often severe. In The Netherlands the summers are cool and delightful but the winters are often severe. The stress and strain of "just living" in Western Virginia

***Vital Statistics in The United States, Part I, 1947—Page 53. Federal Security Agency, Public Health Service.

*A personal communication.

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and The Netherlands thus occurs in the winter.

The above climatic conditions show that if severity of climate is the determining factor of a possible greater incidence of dementia praecox in one season than the other, then the admissions to Eastern State Hospital (which comprise residents of the central and eastern portions of the state) should be those born in the time of stress (the summer) for that region. Also the admissions to Western State Hospital (which comprise residents of the western part of the state) should be those born in the time of stress (the winter) for that region. It was felt that these two opposite climatic extremes might account for the 50% distribution of dementia praecox patients having births in summer and winter. That is—an increase in dementia praecox in western Virginia in winter is compensated by a decrease in dementia praecox for eastern Virginia. An objection might be raised by some at this juncture, that the patients may not have been born in these regions. In many states it is true, the population is not static—consisting of groups on the move for something better. But in Virginia it is a singular fact that a tremendous pride exists in the state. The people are quite contented, thus making their homes where they are born. For that reason these results can be considered fairly accurate for this particular state.

For Eastern State Hospital, the figures show those born in the winter months number 160. Those born in the summer, number 162. This is 50% for each period. Western State Hospital shows figures revealing 210 patients to be born in the winter months and 206 patients to be born in the summer months. This is again 50% for each period. These figures are in the same proportion as the original over-all statistics.

In a final gesture to determine if climatic conditions at birth contribute to the development of dementia praecox, it was decided to review the birth statistics at the colored hospital (Central State Hospital). It was felt that the colored group represented a race which might be more effected by climatic conditions because of being a transplanted race and because of a hereditary background climatically different from that of the white race.

B. Malzberg[4] states that the colored race in this country averages about twice as many (51.1%) cases of dementia praecox per 100,000 population as does the white race (25.6%). Those born in Virginia during the winter months numbered 233 or 47.6%. Those born in the summer months numbered 256 or 52.4%. These results are again so close to the average of 50% as to indicate that the month of birth is relatively unimportant as a contributing factor in dementia praecox.

CONCLUSION: An attempt has been made to see if the month of birth (or the winter season) was important in the subsequent development of dementia praecox as was originally stated by J. de Sauvage Nolting.

1. The collective statistics for the four Virginia state hospitals show 50% of births of dementia praecox patients occur in the winter. 50% likewise occur in the summer.

2. Statistics for the subclassifications of dementia praecox revealed the same percentage for the winter months.

3. Individual statistics for two hospitals serving different climatic regions were obtained. Again 50% of the births occurred in the winter and 50% in the summer. Thus climatic conditions per se at birth (or at conception) seem to have no bearing on the development of dementia praecox.

4. Individual statistics for the colored hospital were obtained. Again, approximately 50% of the births occurred in the winter and 50% in the summer.

As a result, this writer feels that the findings of J. de Sauvage Nolting are not substantiated. He feels also he can state with assurance that the incidence of dementia praecox bears no relation to the individual birth dates of the patients nor to the seasons of the year in which the dementia praecox patients are born.

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PUBLIC HEALTH

The Bureau of Communicable Disease Control
Virginia State Department of Health

Progress of Fourteen Years

The history of public health is intimately connected with the names of men who have aided its advancement. This page, where monthly morbidity has been reported consistently, has been close to the heart of Dr. L. J. Roper, who died June 12, 1951. Dr. Roper was associated with the State Health Department for fourteen years, the last five years of which he was commissioner of health. A study of the following record of these fourteen years gives a picture of the progress made during this period.

CASES AND DEATHS OF LEADING COMMUNICABLE DISEASES 1937 AND 1950

VIRGINIA

Disease	1937 Cases	1937 Deaths	1950 Cases	1950 Deaths
Brucellosis	36	2	67	0
Diphtheria	1,294	89	192	0
Measles	8,825	82	2,959	7
Poliomyelitis	68	14	1,200	62
Rocky Mt. spotted fever	54	9	76	2
Typhoid and paratyphoid	579	57	88	4
Whooping cough	4,324	238	3,293	48

There is no accurate yardstick for measuring the progress of public health work. Emphasis in Virginia is now being placed on the reporting of prematurity. This is evidenced by the new birth certificate. It is not easy to measure the progress made in rheumatic fever, cancer and alcoholic rehabilitation, where comparatively new and actively functioning public health clinics are being operated. With the cooperation of all Virginia physicians, it is hoped that the next fourteen years will continue to see the health department meeting the challenges as they occur and assisting the people of this Commonwealth to be happier and live longer.

MONTHLY MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	June 1951	June 1950	Jan.-June 1951	Jan.-June 1950
Brucellosis	7	5	33	31
Diarrhea and Dysentery	222	203	1,076	1,590
Diphtheria	5	10	68	93
Measles	2,579	486	12,792	2,124
Meningitis (epidemic)	12	11	82	81
Poliomyelitis	6	6	21	21
Rocky Mt. spotted fever	12	13	20	17
Scarlet fever	27	51	669	526
Tularemia	1	0	21	18
Typhoid and paratyphoid	4	1	23	29

Urinary Infections.

A new teaching film, "URINARY INFECTIONS-Bacteriology, Pathology and Treatment", directed by Dr. Grayson Carroll, Associate Professor of Urology, St. Louis University, in collaboration with Dr. Victor F. Marshall, Associate Professor of Surgery (Urology), Cornell University Medical College, has just been completed, it was anounced by Joseph P. Hackel, Medical Film Guild, Ltd. producers of the picture.

The film had its initial showing in the Scientific Program of the annual convention of the American College of Physicians in St. Louis. It was produced under a grant for post-graduate instruction by the Nepera Chemical Co., Inc., and is available without charge for groups of physicians, hospitals and medical schools from the Medical Film Guild, Ltd., 167 West 57th Street, New York 19, New York. Projection service, including projector, operator and screen. also are available without charge upon request.

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MENTAL HEALTH

JOSEPH E. BARRETT, M.D.
Commissioner, Department of Mental Hygiene and Hospitals.

The Guidance Center and the Community*

Guidance centers (sometimes spoken of as mental hygiene clinics) have now been in existence for over 40 years. The first such center was established for a rather limited and specialized purpose, namely, service to a juvenile court. But, as the field of mental hygiene has grown and developed, so have the goals and functions of these centers which are the most practical present-day expressions of this development. These changes have brought the centers closer to the life of the communities in which they work. If a guidance center is adequately to fulfill its role, it must truly become a community agency. As such, it has a threefold function as a medical organization, as a social agency, and as an instrument for public health.

Let us first think of the center as a medical agency. But in so doing we must remember that its other functions make it necessary to avoid the tendency that physicians and hospitals unfortunately have toward isolation from the community. We must also keep in mind that it deals with minor mental pathology rather than with major mental disease. One of the great contributions that the clinics have made to psychiatric practice is the concept of a team approach, a concept which has been in the course of adoption by hospitals for the mentally ill only during the last decade. In the guidance center, a physician (who is a specialist in psychiatry), a clinical psychologist and a psychiatric social worker labor jointly for the welfare of the patient. Each studies and handles his own phase of the situation, but the three pool their information and plan their joint endeavor as equal members of a team in which the psychiatrist is captain. The medical functions are, of course, diagnosis and treatment. It is not always easy to keep a balance between the two. On the one hand, community demands that additional patients be seen promptly are often so insistent as to make extensive diagnostic work crowd out the time needed for satisfactory treatment. Yet, without adequate treatment diagnosis loses most of its value. Moreover, psychotherapy is necessarily slow and time consuming. On the other hand, enthusiasm for therapy may lead the staff of a center into an "ivory tower" or therapeutic work in which they see so few individuals that real service to the community may be limited.

Some guidance centers limit their services to children, while others are "all purpose" and help patients of any age. It would seem that smaller cities can justify the expense of a clinic only if it serves the psychiatric needs of the community as a whole. The child guidance clinic, dealing only with children, is so highly specialized as to fit best in the larger cities or in places where there are other psychiatric resources.

As a social agency, the guidance center is in a position to work cooperatively with many organizations in the community. It can not only be helped by the other social agencies, but can also be mutually helpful to them. An easy interchange of patients and clients who need one service or the other should always be possible. Those who are helping people to solve their personal problems have a large common field of coverage even though they approach their work from different points of view. Each can aid the other. Another example of this is the help which the guidance center can give in the professional develment of the staffs of the social agencies through such media as case conferences, seminars, lecture courses, etc. The clinic can likewise be of service to the schools of a community. This, again, is mutual. The schcol wishes assistance with children whom they find to be emotionally upset, while the guidance center can work most effectively only with the help of the schools because they exert such an important influence in the lives of children. Service may also be given to a juvenile (and occasionally to an adult criminal) court. The task here is not one of determining sanity or insanity, but rather one of helping the judge to understand the personality of the individual before him so that he will be better prepared to make a wise disposition of that person's future. It is, of course, only those judges who see their functions as therapeutic rather than punitive who want such help.

*Article prepared by Dr. Gilbert J. Rich, Psychiatrist-Director, Roanoke Guidance Center.

Finally, as a public health agency the guidance clinic has an educational function. The task of helping people to attain and to maintain good mental health is far too large for all the psychiatrists, psychologists, psychiatric social workers and clinics that we now have in this country or are likely to have for many years. The job can only be approached by helping others to undertake a part of the task. These others are the persons who work with people. They include physicians, public health nurses, social workers, teachers, school administrators, clergymen, lawyers, judges and many others. Educational work is directed towards helping them not only with the particular psychological problems with which they meet, but also toward developing a better understanding of mental health both in general and as it applies to their special fields. Educational work in mental hygiene has eventually to be carried to wider groups in the community, such as women's clubs, parent-teacher associations, service clubs, etc.

To summarize, the task is essentially one of helping people to live happier and more useful lives, for such is the essence of mental hygiene. It is a large task, far too large to be accomplished by a clinic acting alone. The guidance center does its part directly by helping those persons most in need of service and indirectly by aiding others to integrate mental hygiene into daily activities affecting other people.

BOOK ANNOUNCEMENTS

Handbook of Medical Management. By MILTON CHATTON, A.B., M.D., Instructor in Medicine, University of California Medical School. San Francisco; SHELDON MARGEN, A.B., M.D., Clinical Instructor in Medicine, University of California Medical School; HENRY D. BRAINERD, AB., M.D., Assistant Clinical Professor of Medicine and Pediatrics, University of California Medical School; etc. Second Edition. University Medical Publishers, Palo Alto, California. 1951. 508 pages. Price $3.00.

Clinical Laboratory Methods. By W. E. BRAY, B.A., M.D., Professor of Clinical Pathology, University of Virginia; Director of Clinical Laboratories, University of Virginia Hospital. Fourth Edition. St. Louis, The C. V. Mosby Company. 1951. 614 pages. With 119 text illustrations and 18 color plates. Price $7.25.

Philosophy for the Common Man. By HEINRICH F. WOLF, M.D., New York. Philosophical Library, New York. 1951. xxxii-189 pages. Price $3.50.

Paul Ehrlich. By MARTHA MARQUARDT with an introduction by Sir Henry Dale. Henry Schuman. New York, 1951. 255 pp. Price $3.50.

Specific remedies are precious few. Quinine for malaria and colchichum for gout are the only good examples that occur to me. Iron as a cure for anemia is not so good. Mercury and iodides as specifics for syphilis left much to be desired. Until the present era, the drugs that we have used were discovered by primitive peoples or have risen from the ranks from folk medicine. About all that scientific medicine has done is to verify the tradition and to purify and standardize the product. Vaccinia is an excellent example of the process. When organic chemistry developed to the stage where structural formulae were understood, and bacteriology to the stage where the causes of diseases were established, the stage was set for chemotherapy. The chief actor was a small Jewish boy who grew up in Upper Silesia. He was born in Strehlen, March 14, 1854. At school he was very good in Latin and mathematics, but had difficulty with other studies, especially with German composition, so that his teachers had trouble in passing him. At the University in Breslau he was interested only in biology, organic chemistry with its constitutional formulae and in histology. In his first term he transferred to Strassburg. Here he became interested in the new aniline dyes. In his third term he returned to Breslau where he came under the influence of Cohnheim and Heidenhain and where he first met Robert Koch. He completed his university studies in Leipzig, continuing his experimenting with dyes and graduating M.D. in 1878 with a thesis that dealt with the value and significance in medicine of staining with aniline dyes. It contained the idea of a chemical binding of heterogeneous substances to the protoplasm, an idea that logically developed into his famous "side chain theory".

At the Koch Institute, Ehrlich was associated with

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Emil von Behring in his epoch making discoveries of diphtheria and tetanus antitoxin. Indeed it was Ehrlich's chemical genius that devised ways of building up the strength of the antitoxins and of measuring and standardizing them so that they could be used clinically. A small Institute in Steglitz was created for him for serum research and control.

When no more bacteria with soluble toxin, like diphtheria and tetanus were discovered, there was no further advance in immunology along established lines. If a body could not be made to make its own antitoxin, the next step for a chemical genius like Ehrlich was to find or make a chemical with specific selectivity for an invading organism that would have little or no effect upon the host. This, Ehrlich proceeded to do. Trypan red was found to have an affinity for trypanosomes and was of some value in treating sleeping sickness. His experiments with the arsenicals is a story in itself. The first thing that he did was to show that the generally accepted formula of atoxyl was incorrect. Beginning with the corrected formula he introduced various radicals here and there. The 418th preparation, arsenophenylglycin, proved of value. Ehrlich continued experimenting. The 606th compound, arsphenamine, or diaminodihydroxyarsenobenzene dihydrochloride, to give it its full chemical name, was patented under the name of salvarsan. Ehrlich predicted great things for salvarsan, but the assistant to whom he turned it over to test medicinally, reported that therapeutically it was not as good as 418. Ehrlich was greatly disappointed and put 606 aside and continued his search. Some two years later, Kitasato, who had worked with Ehrlich at the Koch Institute, sent one of his pupils, Dr. Hata, to work under Ehrlich. In the meantime the spirochete pallida had been discovered and Dr. Hata had been engaged in Japan in producing syphilis in rabbits. Ehrlich put Dr. Hata to testing 606 again and he soon brought in the report that it was wonderfully efficacious.

The story of the man who stuck to this long trail, vital staining, the side chain theory of immunity and chemotherapy is told in a new biography by Martha Marquardt, who was Ehrlich's secretary for the last thirteen years of his life. She has told the story well and has embellished it with a lot of interesting photographs of world renowned scientists who were pupils of Ehrlich from time to time.

M.P.R.

Physical Examination in Health and Disease. By RUDOLPH H. KAMPMEIER, A.B., M.D., Associate Professor of Medicine, Vanderbilt University School of Medicine; Chief of the Medical Outpatient Service, Vanderbilt University, Nashville, Tennessee. Philadelphia, F. A. Davis Company. 1950. xx-821 pages. With 550 Illustrations, 1 in Color. Cloth. Price $8.00.

The initial training a medical student receives in physical diagnosis depends largely on the skill and patience of his instructors, the amount of supervised exposure to normal and abnormal physical findings, and finally upon the adequacy of his textbook. Numerous textbooks are available, few are adequate, but especially good is the one herein reviewed.

The author has directed his book primarily to the medical student although it contains much of practical value to practitioners of medicine. He has properly divided his chapters to a discussion of each system in the normal with complete instruction on the correct methods of physical diagnosis followed by a description of physical findings in many diseases. The text is graphically emphasized by many excellent photographs which are more impressive than the usual drawings or sketches.

Whenever possible the author gives the anatomy, physiology, and/or pathology involved to explain the physical findings in health and disease. This feature together with the many illustrations make this book far above average in its value as a text in physical diagnosis.

The reviewer highly recommends this book to the student of physical diagnosis as a text book and he will later find it a good reference when he studies disease states. Any doctor who is in practice will benefit by its use as a review of basic fundamentals in physical diagnosis and as a source of information concerning physical findings in many common diseases.

W. H. H., JR.

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EDITORIALS

Miss Agnes Vanculin Edwards

AT a called meeting of the Council, Wednesday, October 11, 1950, Hotel Roanoke, Roanoke, Virginia, Dr. C. Lydon Harrell, President, Medical Society of Virginia, suggested that, in view of the long and faithful service of Miss Agnes Edwards to this organization and its publication, some recognition might be made of her life and that of her father in the Virginia Medical Monthly. Upon this suggestion, Dr. James P. King of Radford, moved that the Editor of the Monthly dedicate an issue to Miss Edwards and her father. This motion was seconded by Dr. W. C. Caudill, Past-President, and was passed unanimously by the Council.

AGNES V. EDWARDS

Dr. Landon B. Edwards (1845-1910), a native of Prince Edward County, Virginia, an alumnus of Lynchburg Military College and of Randolph-Macon College, and graduate in medicine of the University of New York (1867), settled in Lynchburg to practice his chosen profession, after an internship of one year on Blackwell's Island. He soon made for himself a leading place in the medical profession. In 1870 he was a founder of the reorganized Medical Society of Virginia and became its recording secretary, a position he held, except for the year 1883, for the rest of his life.

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When Dr. Edwards moved to Richmond in 1872, he began teaching at the Medical College of Virginia. Later, when the University College of Medicine was started, he transferred his allegiance to that institution, and became at various times professor, clinical professor and chairman of the medical faculty.

In 1871 Dr. Edwards married Nannie Pettyjohn Rucker and to them were born four children: Katherine R., Charles M., Agnes Vanculin, and Landon B. There is another child, not listed in *Who's Who*, and that is the VIRGINIA MEDICAL MONTHLY. He started the MONTHLY as a private enterprise in 1874, and it grew up with the family. For years the English basement of No. 106 W. Grace Street, where Dr. Edwards kept office and published the MONTHLY, was a landmark, just as was a similar office basement further east on Grace Street where the late Dr. William H. Taylor held forth. A passer-by in the leisurely Richmond of the past century could see the occupants at work almost any hour night or day.

Upon the death of Dr. Landon B. Edwards, his son, Dr. Charles M. Edwards, inherited the MONTHLY. When the Medical Society of Virginia bought the journal in the Fall of 1919, they bought Miss Edwards with it. She worked half time November and December 1919 and full time beginning January 1920. The MONTHLY has had few homes and few bosses. Upon the founder's death in 1910, Dr. Charles M. Edwards managed it. When he went to war Miss Edwards took over and has managed it ever since. In 1919 it moved next door to 104½ W. Grace Street and in September 1932 it moved to the Richmond Academy of Medicine Building. Several months ago it moved to 1105 W. Franklin Street, a building that was purchased by The Medical Society of Virginia.

It is hard to date accurately Miss Edwards' connection with the MONTHLY. It began by her substituting for Dr. Landon Edwards' secretary, Miss Ida Thompson, at various times when the latter was sick or on vacation. Officially she was elected Business Manager and Executive Secretary-Treasurer of the Medical Society of Virginia in 1924. The MONTHLY has been the official organ of the Medical Society since its beginning. For a while, between 1896 and 1918, it appeared as a SEMI-MONTHLY, and when the American Medical Association met in Richmond in 1881, it was published daily.

There is nothing about the MONTHLY or the Medical Society of Virginia that Miss Edwards does not know. If you ask her a question, she will answer you right off and then get the minutes or the transactions and show you. We doubt if any one knows the membership of the Society as well as she does. She herself is well known throughout the nation. Wherever we go, someone connected with a medical journal is sure to ask "How is Miss Edwards". She is a charter member of the "Medical Society Executives Conference" and was a member of its executive committee in 1947.

Miss Edwards has been identified with the VIRGINIA MEDICAL MONTHLY so completely, that many believe she has no other interests. This, however, is not so. She is an alumna of Randolph-Macon Woman's College and an active worker in Centenary Methodist Church. She is also a member of the Altrusa Club. Even here she is characteristically efficient. We have tried to find a good joke on Miss Edwards, but even her fellow Altrusians can not tell us one on her.

It would be hard to visualize VIRGINIA MEDICAL MONTHLY without the guiding hand of Agnes Edwards. Her constant devotion and faithfulness have earned her a place in our hearts and we take this opportunity of expressing to her our great appreciation.

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A Review and Analysis of the Report of the Hoover Commission on Federal Medical Activities

YOUR editor confesses that he is confused by the propaganda of the Citizens' Committee for the Hoover Report. Accordingly he wrote to the A.M.A. Headquarters for clarification. Here it is.

The Hoover Commission on Organization of the Executive Branch of the Government transmitted its recommendations on medical activities to the Congress in March 1949, based on two reports received from its Task Force on Federal Medical Services in January and February 1949.

The Task Force recommended that three broad divisions be established in a new United Medical Administration within the Executive Department:

(1) Medical care

(2) Public health

(3) Research and training

It further recommended that the medical functions of the Veterans' Administration in toto, all general and station hospitals of the Armed Forces with minor exceptions, the Public Health Service, and St. Elizabeths Hospital, Washington, D. C. be transferred to the United Medical Administration. It excluded from transfer the hospitals of the Bureau of Indian Affairs and of the Bureau of Prisons, the United States Soldiers' Home, the Naval Home, one medical center for each of the Armed Services, outlying military station hospitals and overseas hospitals and small hospital functions incident to the operations of such agencies as the Tennessee Valley Authority and the Atomic Energy Commission.

The Commission adopted these recommendations and transmitted them to Congress. In addition the Commission recommended that

(1) An advisory board consisting of the Surgeons General of the Army, Navy and Air Force, and the Administrator of the Veterans' Administration be appointed to assist the Administrator;

(2) Congress define the beneficiaries entitled to medical care from the Government and prescribe how this care shall be given, and

(3) The present inconsistency in policy between the Federal hospital construction program and Federal aid to non-Federal hospitals under the Hill-Burton Act be ended.

These recommendations were made "to unite the functions now in five major agencies so as to eliminate overlap, waste, and inefficiency." The Task Force, in conducting the studies prior to the issuance of its report, found the existing federal medical program was "devoid of any central plan" and concluded that "there must be over-all planning and this in turn requires first a clear definition of the extent of the responsibilities, and second an organization appropriate to carry out the commitment." It found the Government "moving into uncalculated obligations without an understanding of their ultimate cost, the lack of professional manpower available to discharge them, or the adverse effect upon the hospital system of the country." For these defects, it blamed principally "the competitive organizaticnal climate in which the various agencies have striven to maintain themselves rather than the agencies themselves." "So competitive," the Task Force reported, "is the environment in which they operate

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that no one agency can now take a Government-wide point of view. As long as this system continues, uneconomical use of medical manpower and facilities will continue."

The Commission recognized that its program could not function unless it was "accompanied by a clear definition by the Congress of the rights and priorities to medical care of all the various classes of beneficiaries." It was thinking particularly of servicemen's dependents and seamen, and most important, of veterans with non-service-connected disabilities, indigent or not. Furthermore, it touches on the complex issue of assumption of Federal financial responsibility for medical care, wherever provided, versus the provision of such medical care directly in Federal hospitals.

The "grave question" was raised whether "the Federal Government should erect an independent hospital system of its own, devoid of community roots, for approximately one-seventh of the total population."

Mr. Hoover and Mr. Manasco dissented "vigorously" from the Commission's recommendation that the Board be "advisory" only and urged instead a board composed of the Surgeons General of the Armed Forces and Public Health Service, and the Medical Director of the Veterans' Administration, with power to determine the policies of the United Medical Administration. Hoover and Manasco said:

> "Because the recommended consolidation of hospitals and certain other medical services is such a revolutionary change from anything heretofore attempted in our Government we, in dissenting, have attempted to find an intermediate step that would overcome an admittedly critical situation and at the same time provide an orderly transition from an outmoded system to an efficient organization. . . . This we feel must be done if the Armed Forces are to meet their obligations in time of peace and have available a trained nucleus of personnel capable of rapid expansion in event of war. . . . *We sincerely believe that unless the Board is given such powers as recommended by us the program will weaken if not destroy military medicine.*" (Italics supplied.)

Commissioners Acheson, Aiken and Rowe dissented from the proposal to set up a new agency and argued instead for a Department of Welfare to include the existing functions of the Federal Security Agency and the hospital activities of the government. As an alternative, they recommended that the Public Health Service be placed in the Department of Welfare even though hospital activities were consolidated in a separate agency.

Brown and McClellan dissented from the recommendation that the hospitals of the Armed Forces be transferred to any new agency on the basis that such a transfer might endanger the quantity and quality of medical and hospital services required by military personnel.

Commissioner Forrestal, in view of his position as Secretary of Defense, abstained from participating at the time of the report.

Thus, of the eleven Commissioners who submitted the Report on Medical Activities Within the Executive Branch of the Government, seven dissented from major recommendations, leaving only four who endorsed the report as a whole.

Bills have been introduced into the 82nd Congress (S. 1140 and H.R. 3688) to carry out the provisions of the Commission's report. In the 81st Congress, S. 2008 was considered by the Senate Committee on Labor and Public Welfare, but was not brought to the floor for a vote.

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The 1951 version of S. 2008 (S. 1140) provides for a Department of Health instead of a United Medical Administration and an Advisory Board consisting of the Secretary of Defense, Administrator of Veterans' Affairs and the Surgeon General of the Public Health Service. Otherwise no important changes are made.

The Hoover Commission's Report was followed by an active public education campaign sponsored by the Citizens' Committee for the Hoover Report and recently a special physicians' committee (The National Doctor's Committee for Improved Federal Medical Services) was named to develop support among physicians and to lend its prestige to the public campaign.

Dr. Walter B. Martin, a Trustee of the American Medical Association, in testifying against S. 2008, in July 1950, on behalf of the American Medical Association, made the following points:

"Any action of the Congress that proposes to place on a more rational basis the federal hospital construction program and to more efficiently utilize federal medical personnel, merits sympathetic consideration. It would seem obvious that the hospital service needs of the Armed Services, the Veterans' Administration and the other agencies of the Federal Government should, to the fullest extent possible, be closely correlated to prevent unnecessary expenditures for additional construction and to assure the maximal utilization of existing facilities and of available medical personnel.

"We are strongly impressed by the Hoover Commission recommendations designed to effect economy in the operation of the several medical services under the Federal Government, provided that economies can in fact be accomplished without sacrifice of the quality of service rendered and without seriously hampering the primary mission of the Armed Forces and the Veterans' Administration.

"A careful perusal of S. 2008 reveals no evidence of how economies are to be accomplished, how the special functions of the several services are to be preserved or how the quality of medical care is to be safeguarded.

"The American Medical Association has repeatedly advocated the consolidation of the medical activities of the Federal Government other than those of the Veterans' Administration and the Armed Forces under a single Department of Health. It has many times pointed out the wastefulness of an uncoordinated hospital construction program and has called attention to the abuse of the hospital facilities of the Veterans' Administration by the admission to those facilities of non-indigent veterans with non-service connected disabilities. Much of the pressure for new construction would be relieved if this abuse were corrected by Congressional action.

"We object particularly to the administrative provisions of the bill. The administrator who is responsible directly and only to the President is given authority to issue regulations without any restraint but that of the President. The Advisory Board, made up of the Surgeons General of the Army, Navy and Air Force, and the Administrator of the Veterans' Administration or their designated representatives, has no real power. The bill provides that the Board will meet at least once each month to consult with the Administrator on policies or proposed policies. It can in no way control the decisions of the Administrator. It is noted that the Public Health Service with its multiple functions and activities has no representation on the Board, nor has the general public.

"The American Medical Association believes that an integrated system of hospitals should be developed for the country as a whole, such as is now developing in many states under the Hill-Burton Act, but that the consolidation of the medical services as proposed in S. 2008 at this time and until such time as careful study has developed more convincing evidence of such a consolidation, would be a risky procedure and fraught with great possibilities of harm.

"The Association is of the opinion that the Congress should proceed slowly in this matter and that any action by the Congress should be preceded by a careful study of

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> **the entire problem, with particular reference to economy, the effect on the quality of medical care and on the execution of the primary mission of the Armed Forces, the Public Health Service and the Veterans' Administration.**
>
> **"It further believes that the principal economy to be accomplished is in the effective coordination of the several hospital systems that would provide for the joint use of available beds and joint planning in the field of hospital construction. It believes that a central hospital board clothed with authority to adjust the hospital program to the needs of the services could gain the end desired without consolidation of professional personnel."**

These remarks are equally applicable with respect to S. 1140, 82d Congress, entitled "A bill to establish and to consolidate certain hospital, medical and public health functions of the Government in a Department of Health."

RECOMMENDATIONS

Recognizing that serious abuses do exist in the conduct of the federal government's hospital system, the Board of Trustees of the American Medical Association recommends:

(1) That a board be established on the federal level to control the distribution of beds among the several federal hospital services and to determine the need and location of proposed new federal hospitals in the continental United States. This board should be made up of recognized authorities in the field of medicine and hospital administration. The Surgeons General of the Armed Forces and the Public Health Service, and the Chief Medical Director of the Veterans' Administration should be consultants to this Board.

(2) That the Armed Forces and the Veterans' Administration maintain their present administrative independence but that they come under the Board with reference to (a) joint utilization of available beds, and (b) construction, expansion and location of federal hospitals in the continental United States.

(3) That the present law pertaining to the hospitalization of veterans with non-service-connected disabilities be reviewed and amended with the purpose of controlling the admission to veterans' hospitals of non-indigent veterans with non-service-connected disabilities.

SOCIETY PROCEEDINGS

Lynchburg Academy of Medicine.

The June meeting of the Academy was ladies' night and a dinner was served at the Boonsboro Country Club. Afterwards a floor show was furnished by Jack Weldon of radio station WWOD.

The only business transacted was the election of a new member, Dr. Joseph Lawson Platt, who was transferred from the Knoxville Academy of Medicine.

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NEWS

Medical Society of Virginia.

Now is the time to be thinking of attending the annual meeting of the Society to be held at the Cavalier, Virginia Beach, October 7, 8, 9 and 10, under the presidency of Dr. C. Lydon Harrell of Norfolk. The program lists papers on a variety of interesting subjects and there will be attractive exhibits, scientific and technical. Representative Hugh D. Scott of Philadelphia will speak following the president's address on Monday evening. Other guests include a speaker from the American Medical Association, and Dr. Winthrop M. Phelps of Baltimore, and Dr. John E. Howard of Baltimore. The usual dinner and entertainment will be enjoyed on Tuesday evening.

A large attendance is expected. Make your plans to be among those present.

American Medical Association.

The recent meeting of the Association, under the presidency of Dr. Elmer L. Henderson of Louisville, was excellent in every detail. Dr. John W. Cline of San Francisco succeeded to the presidency, and Dr. Louis H. Bauer of Hempstead, N. Y., was chosen president-elect. Dr. Oscar Hunter of Washington, D. C., was named vice-president. Dr. George F. Lull and Dr. J. J. Moore, both of Chicago, were re-elected secretary-general manager, and treasurer, respectively. Dr. Walter B. Martin of Norfolk was re-elected to the board of trustees for a term of five years and Dr. David B. Allman of Atlantic City was named for a three-year term to succeed Dr. Bauer, who was named president-elect. The 1952 annual session of the Association will be held in Chicago.

The American College of Chest Physicians

Held its 17th annual meeting at the Ambassador Hotel, Atlantic City, New Jersey, June 7 through 10, with a registration of 1040. On June 9, at the administrative session, the following officers were elected for the coming year: Dr. Chevalier L. Jackson, Philadelphia, President; Dr. Andrew L. Banyai, Milwaukee, President-Elect; Dr. Alvis E. Greer, Houston, and Dr. William A. Hudson, Detroit, Vice-Presidents; Dr. Minas Joannides, Chicago, Treasurer; and Dr. Charles K. Petter, Waukegan, Ill., Assistant Treasurer.

At the Convocation ceremony held on June 9, 109 physicians received their Fellowship Certificates. Oral and written examinations for Fellowship in the College were given to 58 physicians on June 7.

Dr. Charles L. Harrell of Norfolk serves as Governor of the College for the state of Virginia.

Dr. Dean B. Cole of Richmond, serves as Regent of the College for the District No. 4.

Medical College of Virginia News.

On June 5 the Medical College of Virginia awarded 298 degrees or diplomas at its Commencement ceremonies at the close of the one hundred and fourteenth session. Ninety-five of these degrees were in medicine, forty-four in dentistry, fifty-one in pharmacy, fifty-nine in nursing, forty-two certificates in physical therapy, and seven certificates in hospital administration. The latter was the first group to complete the new eighteen-month's course in hospital administration.

Dr. Randolph H. Hoge, professor of gynecology, and Dr. A. B. Gathright, Jr., assistant professor of clinical medicine, gave a series of lectures on cancer from June 4 to June 9 throughout the state of Montana. The talks were sponsored by the Montana Medical Association and the Montana State Board of Health.

On May 21-25 a course in polio treatment, "Nursing Care of the Poliomyelitis Patient", was held at the Medical College of Virginia. The training course, sponsored by the Richmond-Henrico Chapter of the National Foundation for Infantile Paralysis, was well attended by nurses from over the state.

In addition to several adjunct faculty promotions, the following major faculty promotions effective July 1 have been announced: For the School of Medicine—Dr. James F. Blades, Dr. William R. Hill, Dr. John R. Massie, and Dr. T. Duval Watts, Assistant Professors of Clinical Surgery.

Dr. Milton Flocks,

Formerly of Arlington, has moved to San Francisco, where he begun a residency in Ophthalmology at the VA Hospital there.

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Dr. William R. Bond

Has been appointed director of clinical research for the A. H. Robins Company, Inc., of Richmond, effective the first of July. He was formerly engaged in medical research for the Schering Corporation and more recently has served as medical director for Charles C. Haskell and Company of Richmond.

Dr. C. L. Outland,

Medical Director, Richmond Public Schools, has been invited to act as a consultant to the Third National Conference on Physicians and Schools to be held in Highland Park, Illinois, in November, under the auspices of the Bureau of Health Education of the American Medical Association.

Dr. Outland will also serve as discussion leader on the subject: Medical Guidance in Physical Education including Athletics.

News from the State Department of Health.

Dr. James J. Kidney assumed his duties on July 9 as Health Officer of the Russell-Wise Health District with headquarters in Norton.

Dr. John P. Ruppe, Health Officer of the Alleghany-Botetourt Health District, will resign at the close of business, July 25.

Dr. J. N. Rose, a former practicing physician at Harlan, Kentucky, has been named as health officer for Pittsylvania County and will be located at Chatham.

The Second Annual Medico-Military Symposium

Will be held at the U. S. Naval Hospital, Philadelphia, Pennsylvania from 22-27 October 1951, with the view of keeping military Reserve Medical Officers of the Armed Forces, Army, Navy and Air Force posted on the latest developments in the Field of Medical Science.

Commodore Richard A. Kern, MC, USNR, Professor of Medicine, Temple University School of Medicine and Chairman of the Symposium General Committee, will preside over the opening session and introduce the speakers.

It is urged that officers make hotel reservations well in advance, since no government housing facilities will be available. The final session of the Symposium will be held Saturday morning 27 October, leaving the afternoon free for officers to attend the Penn-Navy football game.

Pan-Pacific Surgical Association.

Doctor F. J. Pinkerton, President of the Pan-Pacific Surgical Association, reports plans well under way for the Association's Fifth Congress and advises doctors to make arrangements as soon as possible to attend this meeting. Dates for the Honolulu congress are *November 7-19, 1951.* The scientific program, which will begin on November 12th and continue through November 16th, will include sessions in all divisions of surgery, with papers presented by topflight surgeons from the Pacific area countries. In addition to attending an outstanding surgical conference, doctors may enjoy a delightful vacation in Hawaii and are urged to bring their families with them, with the assurance of luxurious accommodations.

The Pan-Pacific Surgical Association has been officially appointed as travel agent for those attending the congress, and it is therefore important that all hotel and travel reservations be made through the Association office, Suite 7, Young Hotel Building, Honolulu, Hawaii.

The Arthritis and Rheumatism Foundation

Is offering research fellowships in the basic sciences related to arthritis. Fellowships will be granted at both the predoctoral and postdoctoral levels. The predoctoral fellowships will range between $1,500 and $3,000 per annum, and the postdoctoral from $3,000 to $6,000. The deadline for these applications is November 15, 1951. Application forms may be obtained by writing the Medical Director, Arthritis and Rheumatism Foundation, 535 Fifth Avenue, New York 17, N. Y.

A Symposium on Peptic Ulcer,

Sponsored by Kanawha (W. Va.) Medical Society, will be held at the Daniel Boone Hotel, in Charleston, October 3, 1951. The meeting is scheduled to open at 1:30 P.M., and adjourn by nine o'clock.

The program will be presented by Drs. Keith S. Grimson, Robert J. Reeves, Wayne R. Rundles and James P. Hendrix, all of Duke University School of Medicine.

There will be a registration fee of $5.00, including dinner which will be served at 6:30 o'clock. All

interested physicians in West Virginia and adjoining states are invited to attend the meeting.

State Board of Medical Examiners.

At its meeting in June, the Board decided to meet next at the Richmond Hotel, Richmond, December the 6th, and to have the examinations on December the 7th and 8th.

Dr. C. L. Riley of Winchester was elected president of the Board, Dr. C. L. Fifer of Alexandria vice-president, and Dr. K. D. Graves of Roanoke was re-elected secretary-treasurer.

Ground Broken for New Institute of Pathology.

Ground breaking ceremonies for the new Armed Forces Institute of Pathology were held July 10, at the Army Medical Center, Washington, D. C. The $7,250,000 structure will take approximately two years to build, and upon completion will fulfill a long felt need in the field of pathology. The building will be completely air-conditioned and will provide approximately 200,000 square feet of working space. The scientifically designed laboratories will be equipped with the most modern scientific equipment.

During the past half century, five different attempts have been made to obtain funds necessary to construct new quarters for the Armed Forces Institute of Pathology, which before unification of the Military Establishment has been known as the Army Medical Museum and the Army Institute of Pathology. It is due largely to the continued efforts of Brigadier General Raymond O. Dart, a medical officer, who served as the Institute's Director from 1946 until his retirement in 1950, that present plans have been realized.

Dr. George B. Lawson,

Roanoke, has been re-appointed by Governor Battle as a member of the State Board of Health for a term ending June 30, 1958.

Dr. R. S. Griffith,

Ninety-year-old Waynesboro doctor, who in 1900 became a member of Acca Shrine Temple in Richmond, has been honored by Union Royal Arch Masonic Chapter No. 2 of Staunton. He is the oldest member of that organization and a large number paid special tribute to him in June. Dr. Griffith has held a number of State Masonic offices.

Dr. Charles H. Brant

Has been appointed chief of preventive medical service in the Richmond Health Department, a position which has been vacant since January 1950. He is a graduate of the Medical College of Virginia in 1946 and is a former resident of Cleveland.

Southern Medical Association.

The forty-fifth annual meeting of the Association will be in Dallas, Texas, November 5-8, under the presidency of Dr. Curtice Rosser of that city. This will be divided into twenty-one sections and the Woman's Auxiliary will meet at the same time. There should be something of interest for every one.

U. S. Vitamin Corporation Buys Arlington Chemical Company.

U. S. Vitamin Corporation, New York, N. Y. has announced the purchase of the Arlington Chemical Company of Yonkers, N. Y. The 72,000 square foot plant, together with a large new building to be constructed thereon, will be utilized to enlarge the services of both U. S. Vitamin and Arlington to the medical and pharmaceutical professions.

Mr. H. B. Burns, President, states that the present and planned new manufacturing and laboratory facilities, together with an additional score of chemists and other scientists, will lead to the introduction of several new Arlington products.

The Arlington personnel will be maintained and expanded under the direction of U. S. Vitamin Corporation. Main offices of U. S. Vitamin will continue at 250 East 43rd Street, New York 17, N. Y.

The Virginia Section, Southeastern Surgical Congress

Will meet September 13-15 inclusive at the Lord Baltimore Hotel, Baltimore, conjointly with the sections from Maryland, West Virginia and the District of Columbia.

The meeting this year will consist of operative clinics in the mornings and scientific papers in the afternoons of the first two days, with scientific papers on the third morning.

Dr. Waverly R. Payne of Newport News is chairman of the Virginia section, while Dr. W. Raymond McKenzie is chairman of the host state section. Chairman of the program committee is Dr. George Brouillet of Baltimore.

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Dr. Harry Lee Claud is chairman of the District of Columbia section and Dr. E. L. Gage, chairman of the West Virginia section, of which Mr. R. J. Wilkinson, Jr., is executive secretary.

Married.

Dr. Robert M. Miskimon and Mrs. Archer Coke Dennison, both of Richmond, on June 9th.

Criss Award Given for Work in Medicine.

Because of their brilliant work in the development and use of Cortisone, Dr. Philip S. Hench and Dr. Edward C. Kendall of the Mayo Clinic were named joint winners of the first Mutual of Omaha $10,000 Dr. C. C. Criss Award for outstanding contributions in the fields of public health and safety. Presentation of the award was made at the meeting of the American Rheumatism Association at Atlantic City on June 9th.

The Dr. C. C. Criss Award was created in 1949 in honor of the founder and Chairman of the Board of Mutual of Omaha, world's largest exclusive health and accident insurance company. It is awarded to the person or persons residing in the United States, Canada, Alaska or Hawaiian Islands, who, in the opinion of the Board of Judges made the greatest contribution to the public health and/or safety each year.

The Award is open to anyone in the fields of public health or safety. It is, therefore, a real tribute to the great advances that have been made in Medicine that the first winners of the prize, second only to the Nobel prizes in cash value, should be won by workers in this field.

OBITUARIES

Dr. John C. King,

Former superintendent of the Southwestern State Hospital at Marion and founder of St. Alban's Sanatorium at Radford, died June 24. He was eighty years of age and a graduate of the College of Physicians and Surgeons of Baltimore in 1893. He was a charter member of the Southwestern Virginia Medical Society and had been a member of the Medical Society of Virginia for more than fifty years, and had been active in society work. Surviving are his wife and four children, one of them Dr. James P. King of Radford, and also a daughter, Dr. Clara King Dickinson of Marion.

Dr. Bathurst B. Bagby,

For many years active in Virginia public health work, died at his home at Edwardsville on July 6th, at the age of seventy-two. He was a graduate in medicine of George Washington University, Washington, in 1904, and practiced medicine for nineteen years. He took up public health work in 1923 and continued in this until his retirement in 1940. After that he served in civil defense work during World War II. He had been a member of the Medical Society of Virginia since 1905. He is survived by his wife and two sons.

Dr. Rufus L. Phipps,

Proprietor of the Dickenson County Memorial Hospital at Clintwood, died at a hospital in Richmond on July 12th. He was sixty-nine years of age and a graduate in medicine from the Louisville and Hospital Medical College in 1908. He had practiced most of the time in southwest Virginia and was for many years a member of the Medical Society of Virginia. His wife and two daughters survive him.